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APRIL, 1934

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VOLUME 19 APRIL, 1934 NUMBER 4

PERSISTENCE OF APPARENT SINUS PAIN AFTER MULTIPLE OPERATIONS

SAMUEL R. SKILLERN, JR., M.D.

PHILADELPHIA

This difficult problem of rhinology is presented with the hope that the discussion will bring to light some thought that may be the germ center of its final solution. I took the liberty of writing to a number of the leading otolaryngologists of the country, and some of their conceptions are as wide and variable as the four winds of the heavens. Following are portions of their letters.

DR. JOHN F. BARNHILL: I have seen a number of cases such as you describe and have experienced the same difficulties you mention in obtaining satisfactory results from medicinal, operative or climatic treatment.

I have observed more than one cause for such persistent pain: (*a*) an incomplete operation, (*b*) a neurotic condition of the patient and (*c*) neuritis of branches of the fifth nerve. It is my view that the last occurs oftener than otolaryngologists have recognized. Undoubtedly, we all have seen patients who have been well, on whom a complete operation has been performed and in whom drainage is good, as you say, yet who continue to feel miserable. They are not neurotic, at least not in the beginning. The pain is clearly not neuralgic, but it does have many—sometimes all—the characteristics of infection of a sensory nerve.

DR. JOSEPH C. BECK: Pain in the head and face associated with sinus disease, particularly following an operation, is one of the greatest problems that the otolaryngologist has to meet. It is also a fact that many patients are operated on unnecessarily or unsuccessfully, secondarily, for the relief of this pain, when in many instances the disease is elsewhere than in the sinuses operated on.

DR. THOMAS E. CARMODY: My opinion is that in the great majority of these cases the pain is due to blockage, by either scar tissue or swollen tissue.

In some cases I have found synechia between the lateral walls or septum and remaining portions of turbinates, severing of which has relieved the pain.

I shall not discuss the cases in which you found pathologic changes in several cells or in one of the main sinuses, but I think that we overlook a small maxillary sinus cell or a sphenoid cell too frequently.

Another cause which I think we overlook is the fact that some of the sensory fiber may be caught in scar tissue, which we know happens in other parts of the body.

DR. GEORGE M. COATES: I am not a firm believer in so-called habit pain, that is, pain persisting after the removal of the cause. It seems to me that that is like accusing a patient of thinking that he has pain when he has not. It may, when exaggerated, account for some of the persistent headaches, I suppose. In general, my opinion as to the persistence of headache or pain is that it indicates either an incorrect diagnosis or an incomplete operation. If the diagnosis is incorrect, then operation has been performed for headache due to a condition other than in

the sinus, which naturally was not relieved by an operation on the sinus, no matter how radical. In cases of this kind it is difficult to make a diagnosis.

The second cause—incomplete operation—can be avoided if a thorough external operation for pansinusitis is performed. Even then, there is a chance that outlying cells may be overlooked or that it is impossible to reach them.

Dr. Lee Wallace Dean: I have reoperated on patients with sinus trouble on whom I had previously operated because of pain, and have usually found somewhere a fibrous web producing a pocket in which there was stasis. Removal of this relieved the pain.

Dr. Ralph A. Fenton: We must have regard for the fact that scars of operation, especially near the vidian nerve, are often fairly painful. Reoperation often makes them worse; besides, it adds scabbing from the overly roomy nose. The notion that pus drying in a scab in the nose already operated on always means an underlying cell ostium is debatable and should be checked by accurate roentgenograms.

Dr. Perry G. Goldsmith: When the pain is due to a lesion of the sinus mucosa, the majority of these cases respond well when one is able to determine where this mucosa is. I do not believe that the pain is due to a cut nerve or to scar tissue, but it may be caused by an unusually placed cell with secretion under some tension, or in rare instances, by a cell developed in the crista galli.

An uncapped posterior ethmoid cell running above or outside the sphenoid has occasionally been found to be the seat of considerable disturbance; this responds readily to simple drainage.

The essential thing in these cases is to make sure that the diagnosis is correct. So many patients have been operated on, and some have had repeated operations; they are the very type to have their minds distorted by any slight modification of sensation, which is easily magnified in terms of severe pain.

Nervous conditions of the abdomen furnish a simple analogy to this condition. In an atypical form of tic douloureux or migraine a series of sinus operations is frequently performed simply because the patient has some pain in the face and edema of the sinusal membrane. An unerupted wisdom tooth or an early carcinoma of the fossa of Rosenmüller are factors which one must keep in mind.

Dr. Hill Hastings: I agree with you that apparent sinus pains after multiple operations are not always due to a neurotic condition. I have always been inclined to think that they are due to scar tissue after-effects of the sensory nerves. I am under the impression—possibly wrongly—that I seldom meet with persistent neuralgic pain in the head in cases of chronic sinusitis prior to operation. It seems to me that such patients are unusually free from pain, considering the extensive chronic infection that has been present for years.

Dr. Harold I. Lillie: My observations lead me to believe that when the ethmoids and turbinates are operated on for insufficient reasons, not only are the symptoms not relieved, but new symptoms are added. I term these cases "second-handed noses," and little can be done for relief.

It has been my experience that in patients with definite and extensive pathologic changes within the nose well directed surgical intervention is seldom followed by these postoperative syndromes of pain.

Personally, I do not care to take over the job of relieving the postoperative syndromes of pain, and I cannot believe that they can be remedied by removing one or two little cells which may have been overlooked at previous operations. Then, too, the last surgeon who tries to relieve the symptoms is usually blamed for all the trouble.

DR. HARRIS P. MOSHER: Residual pain after operation is due to cells which are unopened. Pain in the cheek is due to interference with the infra-orbital nerve or its branches. Pain after operation not due to these causes is probably due to some other factor than the sinuses, and I doubt if a patient of whom this is true should have been operated on.

DR. WILLIAM V. MULLIN: I am a bit tired of seeing noses and sinuses that have been overoperated on. This experience forces me to conclude that in too many instances they are operated on first with a hope of giving relief from vague symptoms, and that, after the operation has failed to do this, they are given the proper study they should have had before.

We are not justified in assuming that all the types of neuralgia are found in neurotic patients. The cause of some types has never been solved, any more than has the cause of epilepsy.

A misplaced cell that is infected sufficiently to cause the pain of which these patients complain is not difficult to detect. Cells have been in the petrous apex since time was, but much more reference has been made to them lately than ever before. Certainly, all do not require surgical intervention when they are infected. Not many years ago patients with sinus pains were to be relieved by injections into the sphenopalatine ganglion, and the allergists promised to solve all cases of migraine. In spite of all the infected misplaced cells and other foci that we otolaryngologists have removed, general physicians have made better progress in helping arthritis than we have.

Unless there is some definite condition in a nose that has been fairly well cleaned out and unless the pain is localized and not disseminated, I should advise against further operative work. This also holds true in regard to reoperating on a painful sensitive nose to get rid of scar tissue or scattered areas of hyperplasia. It is surprising what helps some of these patients when they get relief from symptoms and are not operated on. I have lived long enough to see the good results of my advice to some patients: "Let your nose and sinuses alone."

If the pain is due to a cut nerve, why does not pain occur in all cases following operation on a sinus? In regard to individual reactions, in some patients scars form, in others keloids and in still others contractures.

DR. ARTHUR W. PROETZ: The real answer lies in a question about which we know little, namely, what causes sinus pain? Almost no study has been made of the mechanism of pain in the nose from a histologic standpoint. When we open a cell, no doubt we frequently affect whatever it is that produces the ache, but perhaps the mere opening and drainage of the cell have little to do with the pain. I am firmly convinced that some severe headaches are caused by diversion of the air currents following operation, which permits the main jet of air to be projected against some sensitive area, such as the branches of the nasal ganglion. It seems likely that some insignificant nerve branch passing through a bony foramen filled with swelling and irregularly contracting connective tissue might cause an incapacitating headache, and as our best operation consists in removal of less than half of the bony wall, the law of averages would give only a 50 per cent likelihood of relief. There are so many noses filled with pus and draining badly which cause no pain that more than simple retention of pus must be responsible for the pain.

DR. ROBERT F. RIDPATH: Unfortunately, all of us have had and will continue to have these cases. They are the bugbear of our specialty. "Why do they have this pain?" can be repeated over and over, with very little resulting illumination. There may be many reasons, some visual, some suggestive and still others unanswerable.

Visual

1. Scar tissue—the result of operative measures—with its meshwork of fibrous tissue, constant contracting tendencies and nerve filaments exposed to unnatural vicissitudes and conditions.
2. Unopened cells. These are more frequently overlooked in the ethmoidal region, but may be, and frequently are, missed in the suprasphenoidal area. In many cases continuous pain, if due to sinus involvement, is caused by an unopened ethmoid cell above the sphenoid sinus or posterior to the maxillary antrum.

Suggestive

1. It has been definitely demonstrated that sympathetic nerve fibers occasionally interwind or surround associated circulatory vessels, either arterial or venous. When these filaments become exposed, either through necrosis of surrounding tissue or by operative measures, pain results.
2. Reflex areas within the nasal nervous membrane associated with the gastro-intestinal, genito-urinary, uterine, ovarian or the deep rectal mucosa. These may take the form of ulcerations, tumefactions, hemorrhoids or fissures, all or any one of which may reflect to the nasal mucosa.
3. Wrong diagnosis. Specialists should not be narrow-minded and think that all pain associated with the upper respiratory tract must essentially be caused by a sinus condition. There are many necrotic, extradural and intradural conditions which may be the causative factor in persistent nasal pain, either before or following operative procedures.

DR. BURT R. SHURLY: After thirty-eight years in the practice of medicine and special work in otolaryngology, I am still unable to evaluate thoroughly the complete significance of symptomatic pain.

My experience in a number of problems of "normal suggestion" in the laboratory of physiologic psychology has led me to believe that the variations found in many people classified as normal give a wide interpretation as to how great pain really is. You are well familiar with the patients of the Krankenhaus belonging to the low grade peasant class who have lived for generations with the sheep, goats and cattle, whose sensibilities are apparently so numbed that many minor surgical procedures and much in the way of technic are carried out without causing any apparent qualm or suffering.

I do not wish to throw the attitude of a skeptic on any genuine case of pain, but your title "The Persistence of Apparent Sinus Pain after Multiple Operations" would of course give great consideration to the state of the individual nervous system, to the hysterical element and to the person who is particularly susceptible to intense feeling.

One of the blessings of mankind is that the memory picture of pain is exceedingly hard to reproduce as a pain and its intensity fades with the passing of the years. It therefore becomes necessary for us as otolaryngologists to classify and evaluate sham and imaginary pain, that manufactured and that intensified to pain which remains. It is only just to complete the physical examination and eliminate all etiologic, diagnostic or reflex nervous and internal concomitant factors.

In these days an increasing number of people are throwing the blame on the physician. This is evidenced by the enormous increase in medicolegal defense. A number of patients go to a specialist with the avowed purpose of "hanging

something" on the surgeon who operated on them, with the intention of bringing a damage suit levying blackmail or at least obtaining a reduction in the bill. In many cases the patients greatly exaggerate their symptoms. With these persons eliminated, I have seen a few genuine cases of recurrent or continued neuralgia or pain after an operation which had not been performed perhaps as completely as possible. However, my experience would show that, with good judgment, experience, skilful technic and a thorough study of the individual case, in only a few cases of several hundred in which a sinus operation had been performed did the patient complain of consequent serious pain, especially pain that could not be relieved by simple restoration of drainage. It is true that sinusitis may and does recur after operation.

The therapeutic nihilists of our specialty and profession have started the slogan "Once a sinus always a sinus." Many of these should have been undertakers, as their optimism and interpretation of facts are those of the physician who spreads his gloom everywhere and believes that his patients are sure to die within another day.

My belief is that recurrent pains of this type that cannot be quickly relieved are not frequent.

Dr. Ferris Smith: The condition is one which has caused repeated annoyance and concern and much speculation on the part of all of us. I believe that the answer may be definite in a considerable number of these cases; in others we can only speculate as to the etiology. I believe that the answer to the question in the majority of cases is contained in the second and third paragraphs of your letter, which read as follows:

"You have, no doubt, had many patients referred to you who gave a history of multiple operations (including the radical group), with but temporary or no relief after operation, yet on examination you find that all the sinuses appear to have been opened and that the nose has apparently excellent drainage.

You treat these cases time after time, and may finally decide to reopen the ethmoid or whatever cell you think is blocked by polypoid mucosa or synechia, but the patient continues to complain."

In the first paragraph you state that all the sinuses had apparently been opened —an impression which we have all had in many cases, only to find that we were incorrect in our observation. In the latter part of the letter you state: "I have seen too many of these cases relieved by opening some small area, usually a deep posterior ethmoid cell that was overlooked at the original operation, and I have noted in nearly all these cases a thin seropustular web somewhere in the nose, which leads me to believe that there is an area of infection which has not been reached."

This has been my observation repeatedly during the past four years. During this period, at the Grand Rapids Clinic we have been managing the ethmoid labyrinth by the direct route, and have been astonished to note the number of cases in which one or more infected cells have been missed after repeated intranasal operations. We were not in the least surprised that the surgeon had failed to open the cell, because its anatomy and situation rendered this impossible. We have been surprised, however, that the intranasal evidences of residual infection or subsequent roentgen investigation did not lead him to suspect the presence of the cell. These cells have been either periorbital, retro-orbital or very thin, slit-like cells along the middle turbinal attachment.

One must go a step further in this particular explanation. Granted that the sinuses have been widely opened for free drainage and ventilation, one has no right to assume that a resolution of the infective and inflammatory process has

occurred in the residual lining membranes as a result of this management, and consequently one cannot rule these out as etiologic factors. On the contrary, we have had many excellent results from complete removal of all lining or bone covering in the involved areas in this type of case and in cases which present persistent local symptoms, despite previous surgical intervention. This, of course, is impossible through the intranasal route and requires the most meticulous care through the direct route. It is my impression that scar tissue, except as it harbors infection, and incarcerated cut nerve ends play a minor part in the problem. I can recall only two cases of pain resulting from incarceration of cut nerve ends in a long period of practice. If it were a real factor here, it would be an equally large factor in all other parts of the body and would be a great deterrent to surgical intervention.

It is my impression that these patients have low grade pachymeningitis in the area immediately covering these cavities and that this is maintained in certain instances so long as the infected bone covering remains. This impression is borne out by the fact that all these patients have headache for a period of from three to fifteen days following the removal of the linings and that this headache is of the type frequently complained of in the cases which you are discussing. Further, we have frequently observed that patients complaining of headache prior to this management complain of increased symptoms immediately following it.

Finally, there is a certain group of cases in which there is definite inflammation of the bone that persists for weeks following complete exenteration of the involved cavities. This has gradually subsided and disappeared in all our cases excepting those in which there was definite osteomyelitis.

We have in the service now a patient whose condition was ably managed by your predecessor on two occasions. He came to us because of persistent intolerable headache. At operation we found three unopened periorbital cells containing pus, a thick infected sphenoidal lining and a small pterygoid pneumatization containing pus. The patient was operated on about seven months ago. He had immediate relief from the acute pain and is enjoying a gradual subsidence of a dull headache, which he said has been present for many years. I believe that this patient had local pachymeningitis and am certain that he had marked infection of the bone, which has finally subsided with the use of bacteriophage locally and systemically.

I believe that one will find the explanation of these difficulties in one's own management of the majority of cases.

DR. FRANK R. SPENCER: It has been my experience that this pain is due to several causes, as follows:

1. Unopened or uncapped ethmoid cells, which are a source of infection and which sometimes have retained secretion, produce pain. Occasionally this has been true of agger nasi cells. This happened in a sister-in-law of our secretary. She had had a number of operations on the sinus, but did not obtain relief until I opened the agger nasi cells on one side.

2. A second source of trouble, particularly in the high dry climate of Colorado, is too free a breathing space following a radical operation, especially on the ethmoid cells, with removal of each middle turbinate. Some of these patients do not have enough mucous membrane left to moisten and warm properly the air breathed in. Medicated oil is required to give relief, even when there is little or no crusting. They have pain in cold weather, but not so often in warm weather.

3. A third cause of pain is some unusual prolongation of a sphenoid or a frontal sinus, with retained secretion. This almost belongs under cause 1.

4. I may add a fourth group—neurotic patients—but I presume from your letter you do not want comment on this group, as we are all familiar with the patient who has trouble in spite of the fact that we have removed every source of infection.

DR. T. H. WEISENBURG (neurologist): The persistence of pains after operations on a sinus is not uncommon. In most of these patients sufficient pathologic changes have been demonstrated either by examination or by roentgenograms to warrant surgical intervention. In a small percentage of cases, however, this has not been so, but facial pains frequently persist following both types of cases.

There are a number of causes for the persistence of such pains:

1. Failure to effect total removal of the pathologic condition.

2. The possibility that the facial pains were not the result of sinus conditions, but had their origin in the implication of branches of the fifth nerve.

3. Pains due to involvement of the ninth nerve, or glossopharyngeal pains. It is not generally recognized that just as there is tic douloureux due to implication of the fifth nerve, there may be pains in the back part of the tongue, throat and neck and in the lower jaw as the result of implication of the ninth nerve.

4. Pains in the face, particularly in the upper part, are frequently the result of implication of the nasopalatine ganglion.

5. Persisting pains following operations on a sinus are frequently of functional origin and constitute a large group of the "atypical facial pains." The experienced surgeon usually dislikes or avoids operating in cases in which the pains are not typical of involvement of either the fifth or the ninth nerve or in which they are not nasopalatine pains. Experience has shown that operations on a sinus and injections of alcohol into the separate divisions of the fifth nerve or the gasserian ganglion or section of the posterior root do not cause cessation of the pains. While in many instances facial pains are undoubtedly of functional origin, nevertheless no patient should be labeled as suffering from a neurosis unless a thorough examination has been made, for usually a neurosis is the result of some psychic or physical change, which can be demonstrated by a thorough neurologic examination.

This leads me to make the broad assertion that surgeons who operate on patients for pain in the face, no matter from what cause—whether the result of implication of the fifth and ninth nerves, involvement of the nasopalatine ganglion or a sinus condition—should always have a neurologic survey made before any radical operation is performed. In support of this position, I have the records of scores of patients with the history of facial pains who have been operated on for sinus conditions in whom the pains have continued and in whom no pathologic changes were found by the surgeon.

The reviewing of these opinions recalls a number of cases of pansinusitis with a history of multiple operations followed by brief amelioration of symptoms. The majority required deeper surgical intervention in the ethmoids, reopening of the sphenoid or maxillary sinuses or radical operations on the frontal sinuses, which gave relief from pain until synechia again walled off these areas and reinfection occurred.

I recently operated on a man, 33 years of age, whose condition was diagnosed as pansinusitis. Owing to the fact that the maxillary sinuses were filled with a heavy purulent discharge, which resisted all forms of treatment, they were cleansed by a radical procedure. Then days later

the ethmoids and sphenoids were opened, and the frontal ostia were rasped. There was marked improvement in the nasal condition as well as in the general health of the patient for six months, when, owing to an attack of influenza, the purulent discharge and other symptoms of sinus disease recurred. The intranasal opening into the right maxillary sinus was found to be closed, and needling was required for irrigation. A heavy purulent mass was washed out of this sinus numerous times, without improvement. Examination following the injection of iodized poppy-seed oil 40 per cent showed the immense frontal sinuses partially filled with polypoid membrane. At a secondary Caldwell-Luc operation, the canine opening in the bony wall was found to be completely closed over by a new bony growth, and the base of the sinus was filled in with new bone and mucous membrane. If I had not been the surgeon, I should have doubted the statement that this sinus had been opened by a radical operation. Within six weeks the opening beneath the inferior turbinate had closed a second time, and the frontal ostia required rerasping. The patient was sent to Florida for the winter, and Dr. Repass reported that the right maxillary sinus is entirely closed off from the inferior nasal passage.

I have reported this case to show how easily some sinuses regenerate their lost tissue and to emphasize the fact that reinfection can and does occur, with a return of pain and other characteristic symptoms, proving to my mind that postoperative pains are often caused by reinfected sinuses.

One must also consider the postoperative changes that occur in the bone, especially in the proximity of the bony canals that transmit the nerves. This is especially true of the infra-orbital canal with its nerve and the foramen rotundum with the vidian nerve. These pains may be compared with sciatica, which the orthopedic surgeon claims is due to a change in the sacro-iliac joint.

The formation of synechia blocking off a cell or involving a nerve must be considered, as must the changes in the blood vessels with their surrounding mesh of sympathetic nerve fibers. Although pain may not be perceived in this sympathetic system, the neurologist states that the sympathetic system is capable of transmitting pain to the sensory nerve or its ganglion:

> There are a number of painful affections about the head and neck that do not conform to any known entity or definite nerve distribution and which are refractory to all treatment. These cases, when improperly diagnosed, lead to useless surgery. When any doubt exists and before major operations are undertaken, cocainization of the sphenopalatine ganglion and the fifth nerve should be performed. If anesthesia results and the pain persists, the case is not one involving these nerves, but probably belongs to the "Atypical neuralgia" described by Frazier.
>
> Glaser gives a résumé of 143 cases of atypical unexplained and unrelieved pain in the face encountered in Frazier's Clinic. The article contains numerous

charts showing the distribution of the pain. It is seen that the pain frequently is felt beyond the trigeminal area, particularly in the neck, shoulder and occiput.

The following conclusions are reached by Glaser: (1) The pain does not follow the direction of the several divisions or branches of the trigeminal nerve, but jumps across anatomic boundaries of the trifacial, even into the neck and arm. (2) The character of the pain suggests disturbance of the sense of pressure. (3) The pain is usually continuous with exacerbations and occasional remissions. (4) The pain is deep seated rather than superficial. (5) Sympathetic disturbances are frequently associated with the pain. (6) The disease is more common in women than in men. Many patients are addicted to the use of drugs. (7) There is no etiological factor common to the majority. Only one, the extraction of teeth, is common to a considerable proportion (15%). (8) Drugs, extraction of teeth, turbinectomies, drainage of sinuses, and all forms of treatment as advocated by cults have been tried and have failed.

The list of futile operations and other therapeutic procedures in these 143 cases are as follows: Injection of alcohol in branches of the trigeminal nerve, 64; cocainization and injection of the sphenopalatine ganglion, 50; extraction of teeth, 48; drainage of sinuses, 48; supraorbital and infraorbital nerve avulsions, 24; other nasal operations, 15; cervical sympathectomy, 12; stripping of the periarterial plexus, 10; subtotal section of sensory root of trigeminal nerve, 11; mastoid operations, 5; pelvic operations, 5.

Dr. Yaskin [1] likened these cases to those of neurologic conditions in the abdomen with multiple scars. He believes that multiple operative treatment is justified, as some pathologic changes are usually found, but that persistence of the pain is due to some as yet undetermined factor.

As a patient of mine so aptly said, "A sinus infection so lowers our morale that it makes cowards of us all." Little pains and aches are magnified into big ones, and those that formerly would have been passed as trivial are thought of as the beginning of some new symptom of sinus disease. Pains in the head, which formerly would have been attributed to some indiscretion, such as eyestrain or overwork, or to the so-called bilious attack are now sinus headaches, and because the patient has been made "nose conscious" by having had a real sinus pain relieved, he believes that every similar pain must of necessity be due to another sinus involvement.

The following is quoted from my brother's [2] writings:

Patients are only too prone to forget the previous severity of their disease and dwell upon some present condition which would have been considered as trivial had they suddenly been transformed from one to the other, but after all it is only human nature and there is not one of us here that would not feel similarly disposed were the personalities reversed and we were the patients instead of the physicians. When a patient undergoes an operation he expects to be cured, yes all cured, and should some slight trace of the old trouble linger or a new symptom

1. Yaskin, Joseph: The Affections of Peripheral Nerves, in Nelson Loose-Leaf Living Surgery, New York, Thomas Nelson & Sons, 1927, p. 618; personal consultation.

2. Skillern, R. H.: The End Results of Radical Operations on the Accessory Sinuses, Ann. Otol., Rhin. & Laryng. **32**:139 (March) 1923.

appear, it is bound to be a source of dissatisfaction, and very real dissatisfaction at that, but if we could only make them realize that the surgical intervention was the means of saving or prolonging their lives or at least rescuing them from a state of chronic invalidism and if they could also be made to understand that the chances of complete recovery are always *inversely* and not directly proportionate to the extent and severity of the operation, the surgeon's task would be far easier than as at present constituted.

So long as there is any evidence of sinus infection, I believe that the obligations of the otolaryngologist have not been fulfilled, and the persistence of apparent sinus pain means further study in conjunction with specialists in other fields until he has found the cause and, if possible, given relief to these unfortunate patients.

RÉSUMÉ OF OPINIONS

A tabulation of the opinions received from the sixteen rhinologists gives the following causes for the persistence of the pain:

Incomplete operation, 7.
Disease elsewhere than in the sinus operated on, 6.
Systemic disease causing symptoms of sinusitis, 5.
Synechia or scar tissue blocking drainage or enmeshing the nerve, and painful contractures, 5.
Direct injury to, or interference with, a nerve, 4.
Operation on the ethmoids and turbinates for insufficient reason, 4.
Individual nervous reaction to pain, 3.
Neuritis of the fifth cranial nerve, 3.
Atypical neuralgia of unknown origin, 3.
Reaction to insult of the periosteum and bone, especially surrounding the foramen, 3.
Injury to the sympathetic plexus, 2.
Diversion of normal air currents within the nose, 1.

SUMMARY OF PERSONAL FINDINGS

The persistence of pain after a radical operation on a sinus may be due to the following causes:

Frontal Sinus
- Failure to remove every vestige of infected material, whether mucous membrane or bone, with the spreading of the infection in the sinus operated on.
- Localized meningitis.
- Abscess of the frontal lobe.
- Infection in another sinus.

Maxillary Sinus
- Injury to the infra-orbital foramen.
- Injury to the dental or infra-orbital nerve.
- Reinfection from overlying sinuses.
- Failure to remove all the infective material from the sinus.
- Infection of an adjacent cell (anterior ethmoid), causing symptoms resembling those of maxillary sinusitis.

Ethmoid Capsule and Sphenoid Sinus

Traumatized periosteum or bone near the foramen.

Direct injury to the nerves or blood vessels, with transmission of the insult to the sensory nerve or ganglion by the enmeshing sympathetic nerve fibers.

Proximity of the meninges and localized meningitis.

Failure to open the long, narrow fronto-ethmoid, the thin, slitlike orbital or the anatomically misplaced ethmoid cell filled with highly toxic material.

The ethmoid capsule and the sphenoid are considered as one, as synechia follows an operation, with walling off of isolated areas of infection.

For all the sinuses a low grade reinfection must be considered, which one is not called on to treat until the area is walled off by scar tissue and the pain recurs.

The causes just given in conjunction with pains in the head from systemic conditions and the atypical neuralgia of Frazier are, in my opinion, the chief factors in the persistence of apparent sinus pain after multiple operations.

PROPHYLACTIC MEDIASTINOTOMY FOR PERFORATING ESOPHAGEAL FOREIGN BODIES

REPORT OF THREE UNUSUAL CASES

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AND
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Esophageal foreign bodies, particularly when they are pointed and embedded against the plane of removal, present almost unsurmountable difficulties to the operator. Even the greatest skill in these cases results in some degree of esophageal laceration, which is usually a superimposed extension of the preexistent perforation.

It is not our purpose to report a great number of cases over a period of years with tabulations of percentages, although we do not deny that statistics serve their purpose. It must be remembered that often these may be applicable for the reporter, but with another geographic site and certain changes in personalities, not only of the surgeons but also of the patients, statistical compilation unrecognizably alters the moral. The following brief reports typify this natural consequence: In China,[1] foreign bodies are more frequently found in the esophagus than in the respiratory tract. An article from Los Angeles [2] records as a primary conclusion that "disks are the commonest forms of foreign bodies occurring in the respiratory and upper digestive tracts" and one from Philadelphia [3] reports that "bones occur in greatest numbers," whereas the foreign bodies most frequently encountered in our experience have been safety-pins. Nor shall we cite cases in which expected complications occurred, but rather we shall emphasize those in which, through no fault of our own or of the patient, expected complications did not take place. The latter type has always reminded us of our prophetic limitations, inherited as doctors of medicine. We hope that the following histories

From the Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine, and the Research and Educational Hospital.

1. Liu, J. H.: Foreign Bodies in the Esophagus, China M. J. **43**:556 (June) 1929. Clerf, L. H.: Peroral Endoscopy, Arch. Otolaryng. **11**:358 (Feb.) 1930.

2. Jesberg, S.: Disks in the Esophagus, Arch. Otolaryng. **11**:210 (Feb.) 1930.

3. (*a*) Jackson, C.: Foreign Bodies, in Piersol, G. M.: The Cyclopedia of Medicine, Philadelphia, F. A. Davis Company, 1932, vol. 5, p. 486. (*b*) Fishman, L. Z.: Mechanism Involving Foreign Bodies in the Postcricoid Narrowing, Arch. Otolaryng. **17**:747 (June) 1933.

with their moral will give us another philosophical problem to consider in our medicoscientific aspirations, the ever present "unknown equation."

There should be no doubt in the minds of competent surgeons that confined nonspecific infections, no matter where they may be, must eventually be connected directly with the outer surface of the body. Whether or not one surgeon decides to wait ten days for the formation of a pyogenic membrane, while another prefers a delay of fourteen days, depends entirely on individual experience. Furthermore, the history of a case, correlated with a complete survey of the patient's status, permits sufficient elasticity in judgment to provide interesting material for debate between consultants. The verdict is sometimes rather unfortunate for the younger surgeon, who usually cannot afford to procrastinate and often appears as an alarmist, whereas the older consultant, if he desires to be contrary, may advise waiting, only to perform the operation finally himself. Given a common definition, fundamental knowledge and experience, two or more different clinical applications of our pseudoscience to an identical situation may each hold forth the promise of success. Tolerance to one's colleague who may differ in opinion on an issue is, therefore, not only advisable but to be hoped for.

Our obvious duty in this report is to emphasize the uncertainty accompanying that period following known perforation of the esophagus, either before or during the removal of the foreign body. The problem resolves itself, then, into the advisability of prophylactic mediastinotomy (cervicotomy).

RÉSUMÉ OF RATIONALE FOR TREATMENT OF PERFORATING ESOPHAGEAL FOREIGN BODIES

We found little information in the literature or textbooks on the important subject of prophylactic mediastinotomy for esophageal perforations. Definition of our aim precludes a review at this time of internal versus external drainage of periesophageal abscesses.

Whereas one of us (F. L. L.) has been decidedly and successfully averse to performing this presumably heroic measure, the Viennese school [4] radically teaches that prophylactic mediastinotomy must always be carried out below the known site of esophageal laceration. The greatest number of esophageal foreign bodies lodge in either the first (postcricoid) or the second natural (aortic) narrowing, so that when the involvement resides within the cervical length of the esophagus,

4. Haslinger, F.: Personal communications in Vienna (F. L. L., 1930; L. Z. F., 1931). Miller, J. W.: Extraction of Foreign Bodies from the Food and Air Passages by Direct Methods, Arch. Otolaryng. **16**:196 (Aug.) 1932. "In perforations of the cervical esophagus, when one is sure of the perforation, it is advisable to do a prophylactic mediastinotomy through the neck, Marschek's method."

laryngologists must consider the aforementioned rational therapeutic measures as a part of their specialty. If thoracic lacerations are the major problem, the cooperation of a thoracic surgeon is unquestionably to be desired. For reasons already mentioned, this need is uncommon, as it has been our experience that the cervical portion of the esophagus is the most usual site.

In discussing a paper by Orton on mediastinitis following esophageal foreign bodies, Clerf [5] stated:

> I am inclined to be conservative in the treatment for esophageal foreign body complicated by mediastinitis. If the mediastinitis developed following esophagoscopy, delay is indicated; if the mediastinitis developed in the absence of instrumentation, esophagoscopy might be indicated.

This statement, of course, is somewhat ambiguous, since Clerf does not indicate whether or not his rationale is based on the knowledge and presence of a foreign body within the esophageal wall, or, what he would do in the event that it had already been removed; and finally, because of lack of definition, the problem of descending mediastinal infection has been left hanging in the air.

Startling enough is the experience of Carmody,[5] who found that when the external operation is performed, practically all of the patients die, and that drainage probably can be established just about as well with suction through the esophagoscope. Here, again, we find dogmatic and misguiding statements. Although his patients may have failed to recover, certainly this is not true of all cases. A possible explanation is that in his particular group the patients were practically dying before he was consulted.

The general confusion as a result of these two contradictory schools was summed up by Orton [6] when he asked:

> What do these cases teach one; what are bronchoscopists to do? They lead me to take the stand of noninterference until the infected area has been walled off, or to place the esophagus at rest by a gastrostomy until such time as the process may be walled off, thereby permitting the thoracic surgeon to operate. Or are we to turn these cases over to the thoracic surgeon immediately at the time of diagnosis?

Peculiarly, he has omitted entirely the vital question of surgical intervention by the laryngologist as a prophylactic measure, and has concerned himself with the less important problem of endoscopy.

A classification suggested by Orton [6] as a guide to rational therapy seems to be post mortem rather than ante mortem:

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5. In discussion on Orton, H. B.: Arch. Otolaryng. **12**:848 (Dec.) 1930. (The article discussed is cited in footnote 6.)

6. Orton, H. B.: Mediastinitis Following Esophageal Foreign Body, Arch. Otolaryng. **12**:635 (Nov.) 1930.

One type perforates at once, and mediastinitis develops, for which I do not think anything can be done. The other type of foreign body is one that perforates possibly by erosion that has been taking time, and in that way has caused a periesophageal condition and might possibly wall off, in which case there is a chronic subacute abscess of the mediastinum which can be drained, as Dr. Imperatori stated, through the esophagus.

How is one to know at the onset, before it is too late, which type of infection the patient is to have? If mediastinitis is going to develop, is it not possible that one may prevent the descent of the infection from the esophagus by placing in its way a mass of resistant scar tissue and leading the purulent material to be formed out from the deeper recesses of the neck to the surface?

In a recent presentation [7] on bronchoscopy and esophagoscopy, one of us (F. L. L.) discussed his conservative view on prophylactic mediastinotomy. Dr. Chevalier L. Jackson of Philadelphia, who was present, quoting the experience of Dr. Chevalier Jackson and himself, agreed with the policy of conservatism as expressed. Although in a great number of instances conservatism may prove successful, one of us (L. Z. F.) is inclined to feel that in many cases there also exists a great danger from a lack of radicalism. The foregoing view may appear obvious, yet we are in complete discord with a recognized school, that of Hajek's clinic at the University of Vienna. A method which is advanced by such an authority must be given an opportunity of trial by competent groups; otherwise when prophylactic surgical measures are not used, failure of patients to recover from esophageal lacerations may be laid at the door of the laryngologist's conservatism.

These controversial ideas are a natural consequence of the inherent uncertainty of prophylactic mediastinotomy, so that the ultimate result is a lack of decisive teaching regarding early lacerations and frank periesophageal infections. Finally, when mediastinal infection does occur in spite of all that has been done surgically, we feel inclined to do as Imperatori [5] suggests, "pray, if one knows how."

THE UNKNOWN EQUATION

The following cases, together with our own, vividly demonstrate the curious sequelae which may occur in cases in which we are entitled to expect severe complications if we wait, yet are pleasantly surprised at the complete failure of the body to react in this untoward manner. In other cases reported the surprise of the surgeon has been almost fatal for the patient.

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7. Lederer, F. L., and Fishman, L. Z.: Bronchoscopy and Oesophagoscopy, Am. Cong. Phys. Therapy, 1933.

REPORTS OF CASES

CASE 1.—One of us (F. L. L.)[8] extracted from the esophagus, with evident laceration, an embedded safety-pin, open against the plane of removal. An uneventful recovery followed. This same patient was sent in again from Elgin State Hospital, a few months later, with another open safety-pin within the postcricoid narrowing, with its pointed branch facing upward. After an unsuccessful attempt to remove the pin had already been made, one of us (L. Z. F.) was called and found that the pin had been pushed down almost to the level of the aortic narrowing, so that pulsations could be seen just below the keeper branch. A safety-pin closer (Haslinger) was successfully used, without first attempting to push the pin farther down, for fear of perforating the aorta. Attempted removal of the closing forceps met with resistance. Disengagement and removal of the forceps showed the pin closed on a portion of the esophagus. Since there was no pin cutter to be obtained, traction on the closed pin resulted in its removal and a laceration of the esophagus, from 1 to 1.5 cm. in extent.

A weak solution of tincture of iodine was applied to the wound, and after twenty-four hours of rest the patient was placed on a boiled liquid diet. Although we daily expected that mediastinotomy would have to be performed, she made an uneventful, afebrile and adysphagic recovery.

It is of interest to note that while the pin was open it remained stationary and showed only the keeper branch from above, but when closed, it moved in a lateral plane to the left with each inspiratory opening of the esophagus, at this instant disclosing its entire length within the lumen.

CASE 2.—Miss S. R. was referred to our service at the Research and Educational Hospital from Elgin State Hospital with a history of having swallowed a box of pins. The first roentgenogram (fig. 1) graphically demonstrated the situation. The intestinal tract was seen filled with over a hundred straight pins, two hair pins and five safety-pins, three of which were open. Three days later, after the patient had passed quite a number of these foreign bodies, the roentgenogram showed only two safety-pins and a great number of straight pins. On June 24, the fifth picture showed the straight pins widely scattered and only one open safety-pin in the descending colon. Later that same day, the patient became violent, requiring her confinement in a strait-jacket, which was secured by means of large horse-blanket pins. Somehow or other, she succeeded in removing one of these and swallowed it. Because we could not obtain a roentgenogram over the week-end, we decided not to interfere without a knowledge of the pin's position. On Monday, June 27, a roentgenogram (fig. 2) revealed the pin, closed and slightly bent within the descending colon, where it remained until passed, open and bent, (fig. 3), with other pins on July 6, 1933, twelve days after its ingestion. One roentgenogram showed the astonishing effect of the intestinal musculature as it was in the process of opening and bending the large safety-pin. The roentgenogram made on July 13 (fig. 4) showed only two straight pins remaining in the lower region of the sigmoid colon. After the patient swallowed the large horse-blanket pin, definite bronchopneumonia developed, from which, after a mild course, she recovered.

8. Fishman,[ab] case 2.

We are safe in assuming that at least one of the hundred odd sharp bodies produced some esophageal laceration. Although no evidence of this ever became manifest, we warn against applying this principle in the management of intractable esophageal foreign bodies. We certainly agree with Jackson [9] that it is not the safest and sanest procedure for the general practitioner, and especially not for the laryngologist, to force these foreign bodies into the stomach. It is not uncommon for us to meet with colleagues who make this the usual rather than the excep-

Fig. 1.—First roentgenogram, June 20, 1933, showing the large collection of foreign bodies within the ascending colon and a scattering of other pins within the transverse and descending colons. The arrows point to the hairpins.

tional method of choice. Our concluding reports will show that foreign bodies less innocuous than pins have necessitated transabdominal surgical removal.

Case 3.—Mrs. B. L., on presentation, showed a roentgenogram which revealed a foreign body at the level of the sixth and seventh cervical vertebrae. The pictures were taken three days (Jan. 12, 1929) after she had noticed, while eating, a sticking sensation deep within the throat. The only symptom was that of slight

9. Jackson, C.: Bronchoscopy and Esophagoscopy, ed. 2, Philadelphia, W. B. Saunders Company, 1927, chap. 18, p. 236.

difficulty in swallowing. Esophagoscopy failed to disclose the site of original trauma. "Watchful waiting" permitted this patient to go along without progressive symptoms of esophagitis. Final roentgen studies showed the foreign body in situ.

This report of esophageal perforation and uneventful recovery reveals a surprisingly reactionless acceptance of a foreign body in the

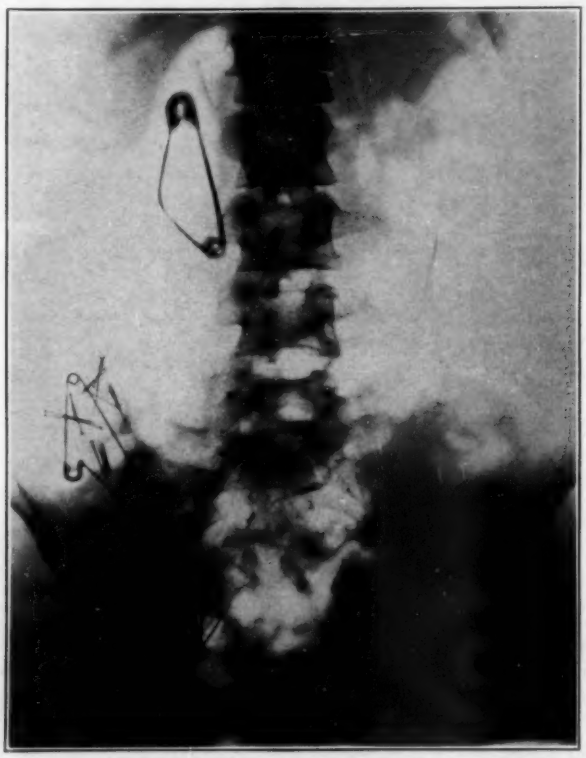

Fig. 2.—Seventh roentgenogram, June 27, showing the large pin in practically the same position, but slightly more bent than on the previous day. The open safety-pin has been turned upside down.

periesophageal spaces after four and one-half years. Imperatori [10] reported the removal of a large Star safety razor blade from the esophagus after it had lacerated the esophageal walls, followed by an uneventful recovery.

The following reports complete our equation and dispel the optimism engendered by the foregoing cases.

10. Imperatori, C. J.: An Unusual Foreign Body, Arch. Otolaryng. **11**:213 (Feb.) 1930.

Case 4.—Mrs. W. L. K. had an aggravated esophagitis, following esophagoscopy because it was suspected that she had swallowed a toothpick while eating a club sandwich. Roentgenograms and this first esophagoscopy failed to disclose any foreign body. One week later, a life-saving incision through the pyriform sinus was performed. About 3 or 4 ounces of foul-smelling, purulent material was aspirated from the upper mediastinum. The abscess drained profusely, and the patient began to recover. One and one-half months later, she noticed pus coming from her mouth, and on Nov. 11, 1930, she came to one of us (F. L. L.), with the complaint of limited motion of her head and ability to swallow best with the head flexed. Examination revealed a draining sinus in the posterior wall of the hypopharynx, to the left of the midline, opposite the middle of the epiglottis. Roentgen studies of the sinus tract following injection of iodized poppy seed oil 40 per cent showed that it communicated with a retro-esophageal abscess in the upper mediastinal region. Bacterial studies revealed fusiform bacilli, spirilla, pneu-

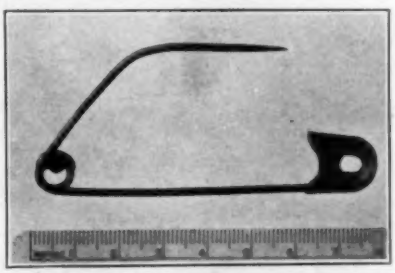

Fig. 3.—The large horse-blanket pin, open and bent.

mococci and threadlike fungi. No acid-fact bacilli were found. Roentgenograms showed the cervical vertebral bodies intact.

Chevalier L. Jackson stated that his prognosis in such cases was that the outcome would be fatal;[11] his opinion coincided with that of Carmody[5] and Orton[6] as already quoted. Because of the insistence by the patient's husband, a physician, on some conservative surgical intervention, intrapharyngeal incision of the sinus and curettage of the cavity were performed on December 12. After a number of exacerbations, the entire process subsided in April, 1931, seven months from the day of onset. The patient returned once more on Jan. 13, 1932, with symptoms of esophageal obstruction, which was shown to be the result of a circular band of scar tissue at the upper end of the esophagus, below which there was also a longitudinal stricture. These are slowly responding to dilation.[12]

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11. This information was given by Dr. Jackson in a telephone conversation to Dr. W. F. Zinn (Baltimore) without seeing the patient, and Dr. Zinn quoted Dr. Jackson as saying that in all his experience he had not seen a single patient recover. This was a fact up to that time, but in a personal communication recently (Dec. 5, 1933), Dr. Jackson stated that he had seen recoveries since then.

12. Lederer, F. L.: Malignant Growths About the Head and Neck, Arch. Otolaryng. **15**:945 (June) 1932.

The bizarre reactions of nature provided Hiller [13] with another point of view on medicine when he found on removing a gangrenous Meckel's diverticulum that the pathologic process had been produced by a rolled tomato peel, shaped like a toothpick. Nutting [14] observed the descent of a thermometer through the gastro-intestinal tract, to find that it had become stationary after a few days. Operation disclosed the thermometer filling entirely the appendical lumen; removal was effected by appendectomy. Swain [15] related our last example of the other half of

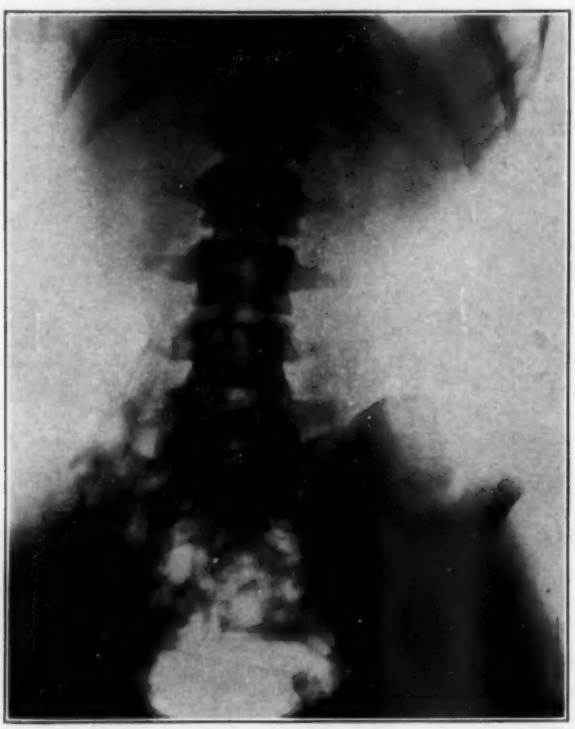

Fig. 4.—Last roentgenogram, July 13, showing only two straight pins remaining in the lowest portion of the descending colon.

life's balance, the case of a 10 month old infant in whom a piece of wire, 3.5 cm. long, eroded the esophagus, passed through into the aorta and produced a false aneurysm. Death took place on the tenth day after onset from rupture of the aorta into the esophagus.

13. Hiller, R. I., and Bernhard, L. A.: Gangrenous Meckel's Diverticulum Perforated by a Tomato Peel, J. A. M. A. **101**:364 (July 29) 1933.

14. Nutting, G. K.: Appendectomy for Swallowed Clinical Thermometer, J. A. M. A. **100**:572 (Feb. 25) 1933.

15. Swain, H. L.: An Unextracted Foreign Body Penetrating the Aorta of a Ten Months Old Infant, Arch. Otolaryng. **12**:544 (Oct.) 1930.

SUMMARY

1. An unusual case of multiple small and large sharp foreign bodies within the gastro-intestinal tract is presented. All of these were passed by rectum without complications.

2. Case histories of esophageal foreign bodies from the literature and from our own experience are presented, the outcome of which has shown that after a diagnosis of esophageal perforation has been made, we have no means of determining whether the conservative or radical form of intervention is to be desired.

3. Safety-pin closers are not always used without danger. The surgeon should be absolutely certain that the point of the safety-pin is not embedded, either originally or as a result of purposeful disengagement, before he attempts to make use of such mechanical devices as safety-pin closers.

CONCLUSIONS

1. We are not advocating routine prophylactic mediastinotomy, according to Viennese concepts; but the fact that the greatest number of patients do not survive periesophageal infections following perforations by foreign bodies, added to the fact that this operation is so little used in America, certainly suggests that many prognoses may be improved by prophylactic mediastinotomy.

2. A plea is made for rational conservatism and radicalism.

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INFECTION OF THE PETROUS BONE

RATIONALE OF TREATMENT AND REPORT OF A CASE

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Since 1930 much has been written of infections of the petrous bone by Profant,[1] Eagleton,[2] and especially Kopetzky and Almour.[3] A satisfactory clinical picture has been worked out, thanks to Kopetzky, so that the diagnosis can be made more easily than in the past. Various operative procedures have been devised, notably by Eagleton, Almour, Voss and Frenckner, whose methods are tolerably familiar to most otologists.

However, as yet the results attained by these workers do not approximate the mortality figures of Perkins,[4] who in 1910 collected ninety-four cases showing Gradenigo's syndrome, with only eleven deaths (about 12 per cent). Treatment at that time was limited to a simple or a radical mastoid operation. Kopetzky's mortality was about 43 per cent (three of seven cases in which Almour's technic was used).

From Perkins' figures it is obvious that surgical drainage of the petrous tip is not always necessary to preserve life, as shown also by cases 7 and 9 in Kopetzky's original series.

The problem is to select the patients who must receive operative drainage of the tip and those who will recover without such drainage.

In Kopetzky's series, after the initial onset of pain in the eye, otorrhea and low grade sepsis, some of his cases entered a second stage, a stage of quiescence, which is coincidental with the advance of the infection into the epidural region about the tip. He warned the observer not to be deceived by the apparent recovery, but to hasten the drainage of the tip before the onset of a terminal meningitis.

Would it not be as logical to assume that the patient was actually recovering and that the pus had broken through into the middle ear by

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1. Profant, H. J.: Gradenigo's Syndrome: With a Consideration of "Petrositis," Arch. Otolaryng. **13**:347 (March) 1931.

2. Eagleton, W. P.: Unlocking of the Petrous Pyramid for Localized Bulbar (Pontile) Meningitis Secondary to Suppuration of the Petrous Apex: Report of Four Cases with Recovery in Three, Arch. Otolaryng. **13**:386 (March) 1931.

3. Kopetzky, S. J., and Almour, R.: The Suppuration of the Petrous Pyramid: Pathology, Symptomatology, and Surgical Treatment, Ann. Otol., Rhin. & Laryng. **39**:996 (Dec.) 1930; **40**:157 (March), 396 (June) and 922 (Sept.) 1931.

4. Perkins, C. E.: Abducens Paralysis and Otitis Media Purulenta, Ann. Otol., Rhin. & Laryng. **19**:692, 1910.

means of the chain of cells through which the infection had first entered the petrous tip? I suggest that in such a case there is a sudden increase in the quantity of pus discharged from the middle ear or from the mastoid wound, accompanied by regression of the pain in the eye and the low grade sepsis.

With that possibility in mind, would not one be justified in waiting until convinced either that the patient will recover spontaneously or that operation is imperative to save life? Granted that such a course adds some risk, it must also be admitted that an operation for drainage of the petrous bone adds an equal or even greater risk to the patient's life, else why the difference in the mortality figures cited? (Kopetzky's figures may possibly be better in a longer series.)

In the event of ultimate operation, waiting has the further advantage of allowing the pathologic changes to become further advanced and the cells softer and more necrotic, thus increasing the possibility of finding a lead to the tip through a broken-down gallery of cells. To me, this seems entirely logical, and I suggest the following points for consideration after a diagnosis of infection of the petrous bone is established following a mastoid operation:

1. While the pain in the eye, fever and otorrhea persist without change, wait for developments as stated in paragraphs 2 or 3.

2. If the symptoms increase in severity or threatening signs ensue (increase in headache, irritability, vomiting, stiff neck or vertigo), at once explore the petrous tip by one of the usual methods, looking first for a fistula.

3. If the stage of quiescence occurs, watch carefully for an increase or decrease in the otorrhea. If there is an increase, indicating rupture into the middle ear, wait for further developments; spontaneous recovery may occur. If there is a decrease, or if no change takes place, exploration without delay is indicated.

As for operation, I prefer an exploratory one, without a preconceived idea of any definite procedure to follow. If a fistulous tract is found, it is far wiser to drain the tip through this than to create an artificial channel through an uncharted region, without landmarks, proceeding by a mathematical rule which disregards any possible anatomic anomalies.

If no fistula or necrotic tract can be demonstrated, then I should prefer using Eagleton's unlocking operation, modified by omitting the greater part of the removal of the posterior buttress, simply removing the anterior buttress and the tegmen, and entering the tip subtemporally.

REPORT OF A CASE

N. W., a girl, aged 6 years, had an earache on the left side for one week following tonsillitis. A paracentesis was performed on Dec. 26, 1931. Signs of mas-

toiditis developed, and the patient was referred to the Methodist Episcopal Hospital, Brooklyn, where a simple operation for mastoid was performed on Jan. 4, 1932. The mastoid was a well developed pneumatic one, with coalescence. On culture a short chain streptococcus was found.

Following the operation the child complained almost from the first day of left retro-orbital pain. She had a low grade fever, her temperature never rising over 100.8 F. The wound continued to drain. A diagnosis of petrositis was made, and on the principles already outlined I decided to await development before interfering surgically. This patient never entered the stage of quiescence. The ocular pain and the fever continued. Twenty-four days after the operation the temperature rose to 101 F., and she vomited once. The pain persisted and was worse at night. The patient showed increased irritability. Her white cell count on the day of vomiting was 11,000 with 77 per cent polymorphonuclears. At this point she should have had an operation for drainage of the petrous bone.

On the twenty-eighth day her temperature rose suddenly to 103.4 F. She had a stiff neck and a positive Kernig sign; the spinal fluid was under pressure and showed a cell count of 2,000 with 70 per cent polymorphonuclears. No organisms were found on smear. Cultures were made at this time which later proved to be sterile. The white cell count was 12,800 with 76 per cent polymorphonuclears. A diagnosis of adjacent meningitis was made (based on the absence of organisms in the spinal fluid) and an exploratory operation was done about an hour after the rise of the temperature (February 1).

On the basis of Kopetzky's original contribution, I expected to find the point of entrance about the eustachian opening and performed a radical operation, but I found no caries at this point. I then explored the area of Trautman's triangle, and here I found a gallery of necrotic cells leading down under the labyrinth.

This chain of cells was explored with a fine curet and at a depth of one-half inch I broke into a small cavity; about 8 to 10 drops of thick green pus welled up from the petrous tip. The channel was enlarged, and a doubled strand of silkworm gut was inserted to maintain drainage. The dura was not opened. A smear showed a gram-positive diplococcus with a capsule, probably a pneumococcus. There was no growth on culture.

The spinal fluid next day showed only 1,000 cells and no organisms.

The patient rapidly improved, her temperature dropped to 100 F., and the spinal fluid on the fourth day showed only 21 cells.

The temperature then began a septic course, and on the sixth day, February 7, rose to 105 F. I opened the lateral sinus and found a clot. This was removed; the sinus was packed off at both ends, and a jugular ligation was done. The patient received a small transfusion the next day (250 cc. of whole blood), and from then on her course was uneventful. She was discharged from the hospital on March 7.

I have seen the patient at irregular intervals since then; the last time was in August, 1933. She has remained well, has not had any symptoms and has a good cavity with slight moisture (mucus). No pus has drained from the ear at any time since her discharge from the hospital.

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LOCAL AMYLOID DISEASE OF THE UPPER AIR PASSAGES

REPORT OF FIVE CASES

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ANN ARBOR, MICH.

Since 1873, when Burow and Neumann first described amyloid tumors of the larynx, cases have been reported in the medical literature. In the first cases reported the tumors were discovered at autopsy, but with the gradual improvement in clinical methods of examination, more cases have been recognized and confirmed by biopsy.

I am inclined to believe and have been led to understand that local amyloid disease of the upper air passages is relatively rare, since in the entire literature there are not many more than fifty cases. Whether this is due to an actual rarity of this rather peculiar pathologic condition or a lack of alertness on the part of the clinician is difficult to determine. One, however, cannot help but think that this particular disease will be recognized more often in the future.

The upper air passages, according to New,[1] seem to be predisposed to local amyloid disease more frequently than any other part of the body. He classified the disease clinically into three types: (1) diffuse subepithelial infiltration, (2) tumor-forming local amyloid deposits and (3) amyloid degeneration of preexisting tumors. These three types are exemplified in the five cases herein reported. The classification used by Seckel to record twenty-six cases and later adopted by New in completing his series for publication is chosen here as a continuation of the work already done (table).

In cases previously reported, males were affected three times as often as females. In my series, all of the patients were males.

The youngest patient was 35 years of age, the oldest, 62. Three of them were between the ages of 50 and 65 years, and the other two were between 35 and 40.

The two chief symptoms accompanying amyloid disease of the larynx are those which are commonly produced by any mechanism which tends

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From the Department of Otolaryngology, University of Michigan Hospital.

1. New, G. B.: Laryngoscope **29**:327 (June) 1919.

to obstruct the glottis, mainly, hoarseness and dyspnea. All of the patients complained of hoarseness, and two of dyspnea. One was so dyspneic that an immediate tracheotomy had to be performed.

The duration of the symptoms varied, the longest being thirty years. One patient (case 3) complained of soreness of the mouth and hoarseness of varying degree for more than thirty years. He did not present himself for examination until his mouth became so sore that he could no longer ingest food. A diagnosis of ulcerative gummas of the tongue was made, and antisyphilitic treatment was instituted. It was not until eleven years later that a diagnosis of local amyloid disease of the tongue, hypopharynx and larynx was made and confirmed by biopsy. The

Classification of Cases According to Seckel's Method

				Location							Pathologic Diagnosis					
Case	Sex	Age	Duration of Symptoms, Yrs.	Tongue	Tonsils	Pharynx	Larynx	Trachea	Bronchi	Number of Tumors	Clinical	Autopsy	Biopsy	Operation	Associated General Pathologic Changes	Comment
1	M	35	14	..	..	..	+	..	..	Single	..	..	+	..	Fistula in anus	
2	M	58	1¼	..	..	..	+	..	..	Single	..	..	..	+	Carcinoma of larynx	Operated on primarily for carcinoma of larynx
3	M	54	30	+	+	+	+	..	..	Multiple	..	..	+	..	Apical abscesses	
4	M	37	2	..	..	..	+	..	..	Single	..	..	..	+	None	
5	M	62	5	..	..	..	+	..	..	Single	..	..	..	+	None	

symptoms of shortest duration occurred in the patient in case 2, who complained of hoarseness for fifteen months and was compelled finally to present himself for examination on account of dyspnea which had been present in varying degree for six months. It is possible for local amyloid disease of the upper respiratory tract to be present for some time before symptoms appear.

The disease was found in the larynx more often than at any other site in the upper air passages, being observed there in the five cases. In only one patient (case 3) did multiple lesions occur. These involved the mouth, tongue, tonsils, pharynx and larynx. None were found in the trachea.

Tumor-forming local amyloid deposits were the predominant clinical type of disease found, being present in three cases. Diffuse subepithelial infiltration was present in one case, while case 2, one of amyloid

degeneration of preexisting tumors, was the only one of its kind in the series.

The characteristic color of the tumors, when examined clinically, was red, with the exception of that in case 3. This tumor, which was of the diffuse subepithelial infiltrative type, was a waxy yellowish gray. It appeared nodular and was firm on palpation. The tumor-forming local amyloid deposits did not differ clinically from the benign and malignant pedunculated neoplasms seen in the larynx. For this reason I am of the opinion that the tumor-forming type cannot be diagnosed correctly without biopsy.

Having made a diagnosis clinically and microscopically of local amyloid disease in the cases reported here, a study of associated pathologic conditions was also made. Two of the patients presented other findings. In case 1 there was an anal fistula of which the patient was not aware until the time of examination. During routine examination in case 3, roentgenograms of the teeth were made. In four teeth there were pathologic changes about the apexes. None of the more chronic diseases, such as tuberculosis, osteomyelitis or syphilis, which are usually associated with general amyloid disease were found.

The origin of amyloid substance is still somewhat obscure. Ziegler [2] stated that local amyloid infiltrations are met with in granulations which are rich in cells, in chronically inflamed tissues and in hyperplastic connective tissue growths. They occur also in tumors in which other degenerative processes are in progress. Case 2 demonstrates this. Both squamous cell carcinoma and local amyloid disease were demonstrated. He also added that tumor-like amyloid masses are occasionally met with in the conjunctiva, tongue, larynx and trachea under conditions in which it is impossible to establish any relationship between them and an inflammatory process. In only two cases was it possible to demonstrate associated pathologic changes of any kind.

Raehlmann [3] has found that amyloid substance has a predisposition for the connective tissue of vessels. In small arteries it is found first between the muscular coats of the media in variable forms which press laterally against the fibers, causing them to atrophy and disappear. In the veins, on the contrary, it is seen under the endothelium of the intima, while in the capillaries it surrounds the lumen completely. Thus it can be readily seen that as the deposits increase in amount they so

2. Ziegler: Virchows Arch. f. path. Anat. **65**:278, 1876.
3. Raehlmann, E.: Arch. f. Augenh. **11**:402, 1882.

modify the normal conformation of the mucosa and submucosa as to create the different clinical lesions already mentioned.

The treatment in the cases which presented single tumor-forming local amyloid deposits (two) consisted of simply removing the deposits perorally. This method was also attempted in case 5, but because of profuse hemorrhage it had to be abandoned. The tumor was later removed by first performing a thyrotomy and then excising the tumor from the cord. Examination of these patients later showed no recurrence. Because of a coexisting carcinoma in case 2, the patient had to be subjected to a total laryngectomy. The only treatment in case 3, of the diffuse subepithelial type, was a complete course of antisyphilitic therapy. This treatment seemed to have some influence on the disease, and tended to alleviate the pain and soreness associated with the ulcerative lesions on the tongue. Whether the ulcers would have involuted spontaneously without antisyphilitic treatment, I cannot say. New[1] stated that he has used fulguration and radium with marked improvement in cases of diffuse amyloid deposits. This was accomplished by first performing thyrotomy. Roentgen treatment has also been mentioned in the literature, but little is known concerning its effect.

REPORT OF CASES

CASE 1.—H. C., a man, 35 years of age, had experienced a choking sensation periodically for two years. He had had persistent hoarseness since the age of 21 years. No history of dyspnea was given.

Examination showed moderate-sized, injected tonsils. Nothing of importance was noted in the nose or ears. The true cords of the larynx were somewhat reddened. A red, smooth, sessile tumescence about the size of a pea was noted on the anterior one third of the right vocal cord. This was attached to its free edge. There was no associated cervical adenopathy.

Medical consultation showed that the patient had an anal fistula. Roentgenograms of the chest showed nothing abnormal. Neurologic examination revealed nothing of importance. The Kahn reaction was negative, and studies of the urine and blood gave negative results.

By indirect laryngoscopy and local anesthesia the tumescence was cleanly removed from the free edge of the cord. Little hemorrhage occurred following this procedure.

The patient convalesced uneventfully and was discharged from the hospital the third day following the operation. He was examined one month later, and only a slight roughening was seen at the point where the tumescence had been attached. His hoarseness had almost disappeared. Figure 1 shows the appearance of the tumescence on the cord.

CASE 2.—J. G., aged 58, a native of Austria, was admitted to the hospital with a chief complaint of difficulty in breathing. Fifteen months previously some lumber had fallen on his right arm. Since that time he had been hoarse and had

complained of a continuous dull aching pain in his throat. The hoarseness had progressed until, on admission to the hospital, he could only whisper. During the last six months he had gradually become dyspneic.

Examination of the mouth revealed oral sepsis and retained roots of teeth. The septum of the nose was deviated to the left. The ears were normal. When the

Fig. 1 (case 1).—Clinical appearance of the larynx.

Fig. 2 (case 2).—The laryngeal lesion, containing both amyloid substance and squamous cell carcinoma.

larynx was examined, a smooth, red, round mass about the size of a hickory nut was seen in the region of the entire right cord, distorting its normal contour. An area of ulceration was found over the free medial aspect of the tumor. Owing to the approximation of the tumor with the contralateral cord, the mobility of both cords was affected somewhat. No glands were palpated in the neck.

Medical consultants were unable to find any evidence of metastasis in the chest. The blood vascular system was sound.

Roentgenographic examination of the chest gave negative results. Detailed studies of the larynx showed a gross mass of soft tissue apparently arising from the posterolateral wall of the larynx and almost occluding the upper glottic lumen.

Fig. 3 (case 3).—The larynx. Note the diffuse subepithelial infiltration of the laryngeal mucosa by the amyloid substance.

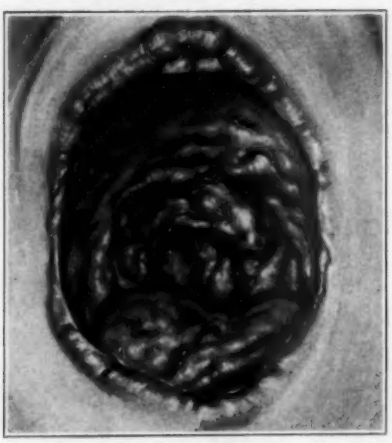

Fig. 4 (case 3).—The mouth, showing diffuse subepithelial infiltrations of amyloid substance.

A biopsy of the tumor showed it to be a squamous cell carcinoma.

Owing to the position and size of the neoplasm, a total laryngectomy was performed, and it was not until this specimen was examined microscopically that an additional diagnosis of local amyloid disease was made.

The patient had a rather uneventful convalescence and was discharged from the hospital a month later, following intensive roentgen therapy of the neck (fig. 2).

CASE 3.—M. H., aged 54, a German, was first examined in the dermatologic section, where a diagnosis of tertiary syphilis of the tongue was made. At that time he had numerous ulcerative lesions on the tongue which closely resembled gummas. He was given an extensive course of antisyphilitic therapy, which consisted of arsphenamine, mercury rubs and iodides. This treatment caused the lesions on the tongue to involute somewhat.

Eleven years later the patient returned to the hospital complaining of soreness of the tongue. He stated that it was difficult for him to speak, and his mouth watered easily.

Examination showed the tongue somewhat fixed in the floor of the mouth, with considerable scarring at the dorsum and its anterior margins. Just opposite the anterior pillar on the left side was an ulcerating area about the size of a dime with a hemorrhagic border. The ulcer was covered with a necrotic, thick pellicle. Small areas of ulceration were observed over the surface of the tonsils. About these areas were numerous, glass pinhead-sized, grayish-white, firm papules. All of the underlying structures of the tongue, pillars and postpharyngeal wall were rather hard and unyielding.

One of the lesions was removed for biopsy, and a diagnosis of local amyloid disease was made. The patient had had three Kahn tests, all of which were negative. Owing to the fact that the lesions in the mouth had improved markedly under antisyphilitic therapy previously, the patient would consent to no other type of treatment; he was given thirteen intramuscular injections of bismuth salicylate and discharged.

One year later the patient was examined in the department of laryngology in regard to hoarseness which had been present for years but which had recently become worse.

The dorsum and edges of the tongue presented a nodular grayish-yellow appearance. The papillae appeared atrophic. When the patient was asked to lift his tongue, it was found to be quite firmly bound to the floor of the mouth. The buccal mucosa, pillars, free surface of the tonsils and postpharyngeal wall were nodular and of the same color as the tongue.

By indirect laryngoscopy the mucosa over the epiglottis and pyriform sinuses was found to be grayish-yellow and nodular, giving a markedly thickened appearance to the epiglottis. The false and true cords were thickened and of the same color. Motion of the cords was only slightly limited by the thickening of the submucosa. The trachea was not involved. On questioning the patient stated that he was in no way incapacitated by these changes in his mouth and throat, except for the hoarseness he spoke of. Because he was not dyspneic and only moderately hoarse, no type of therapy was recommended. The patient is easily accessible and so can be checked up frequently if need be (figs. 3 and 4).

CASE 4.—R. O., a man, aged 37, complained of hoarseness which had been present for two years. He stated that the difficulty with his voice would appear suddenly, last a variable period and then disappear. This symptom had occurred several times.

When examined he was found to have a septal deviation to the right. When the larynx was examined, a small, rounded, smooth, dull red tumor about 3 mm. in diameter was found on the middle one third of the right vocal cord. The tumor seemed to be attached to the free border of the cord and was thrown into vibration by phonation. No other findings were evident on physical examination.

Fig. 5 (case 4).—The larynx, showing the tumor-forming local amyloid deposits.

Fig. 6 (case 5).—Clinical appearance of the laryngeal lesion.

A clinical diagnosis of laryngeal papilloma was made, and the tumor was removed by direct laryngoscopy under general anesthesia. Microscopically, the tissue was found to be affected by local amyloid disease.

Following the removal of the tumescence from the larynx, the patient's hoarseness disappeared (fig. 5).

Case 5.—H. B., aged 62, stated that five years previously he had suddenly become hoarse. After a short time this disappeared. Following the first attack others of a similar character occurred. Three months previous to his entrance

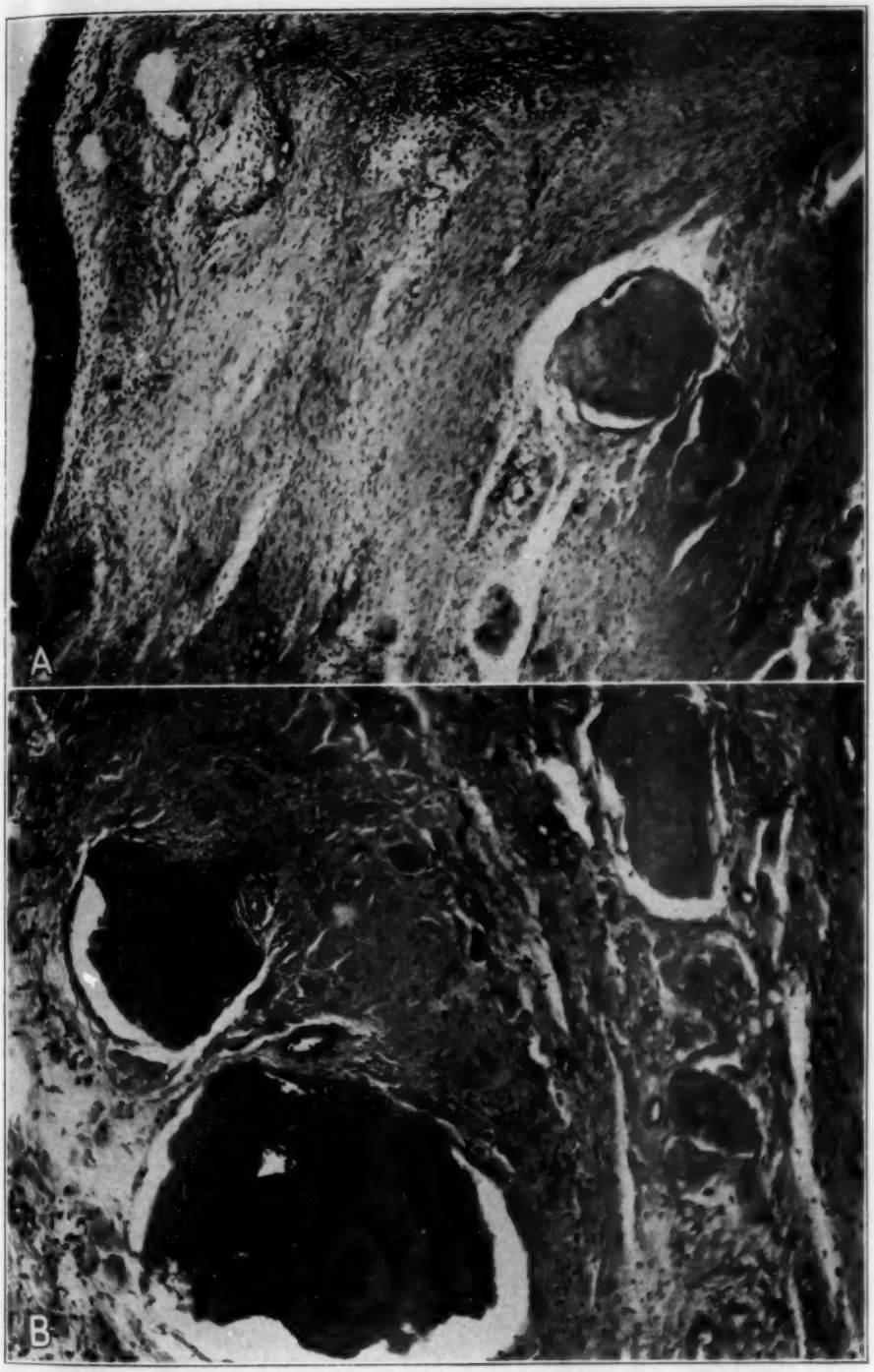

Fig. 7 (case 5).—*A*, low power photomicrograph of the laryngeal polyp, showing amyloid tissue concretions in the submucosal inflammatory tissue. *B*, higher power photomicrograph of the amyloid tissue concretions in *A*, with numerous smaller hyaline masses developing throughout the inflammatory connective tissue. All of these hyaline masses arise in blood vessels. The smaller, less dense ones stain as connective tissue hyaline; the larger, more dense ones give the amyloid reaction with cresyl violet, methyl and gentian violet and other metachromatic amyloid dyes.

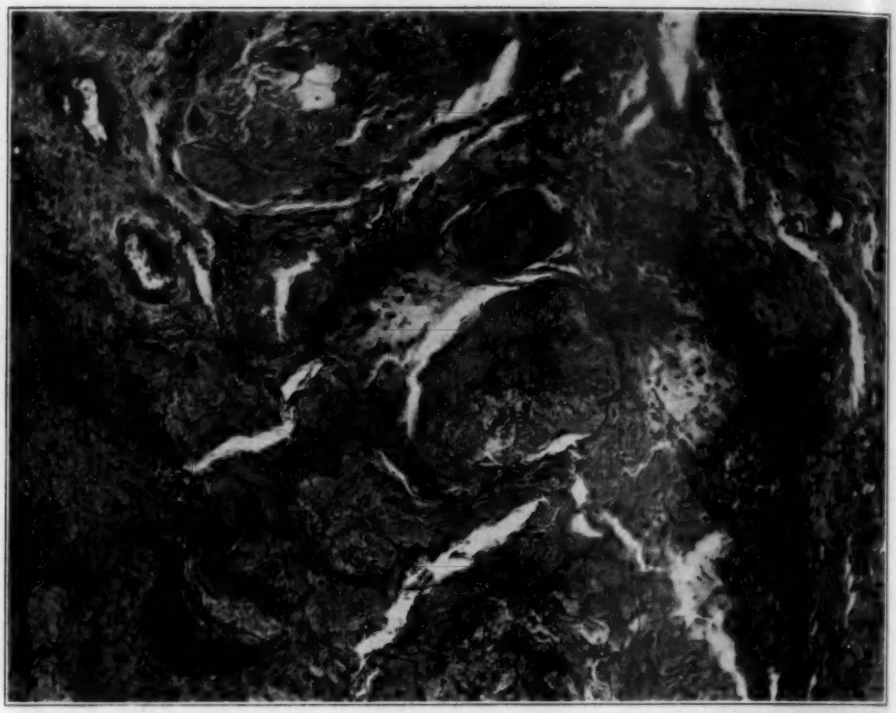

Fig. 8 (case 5).—Medium high power photomicrograph of the hyaline and amyloid concretions in the laryngeal polyp. The hyaline masses represent transformed blood vessels.

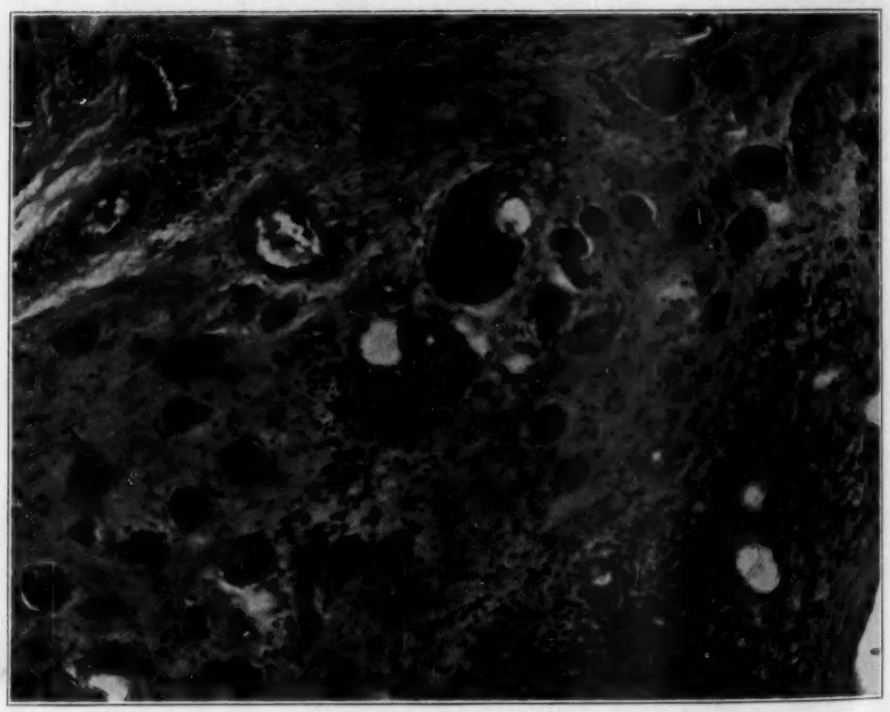

Fig. 9 (case 5).—Development of hyaline and amyloid tissue concretions in the laryngeal polyp from the small blood vessels.

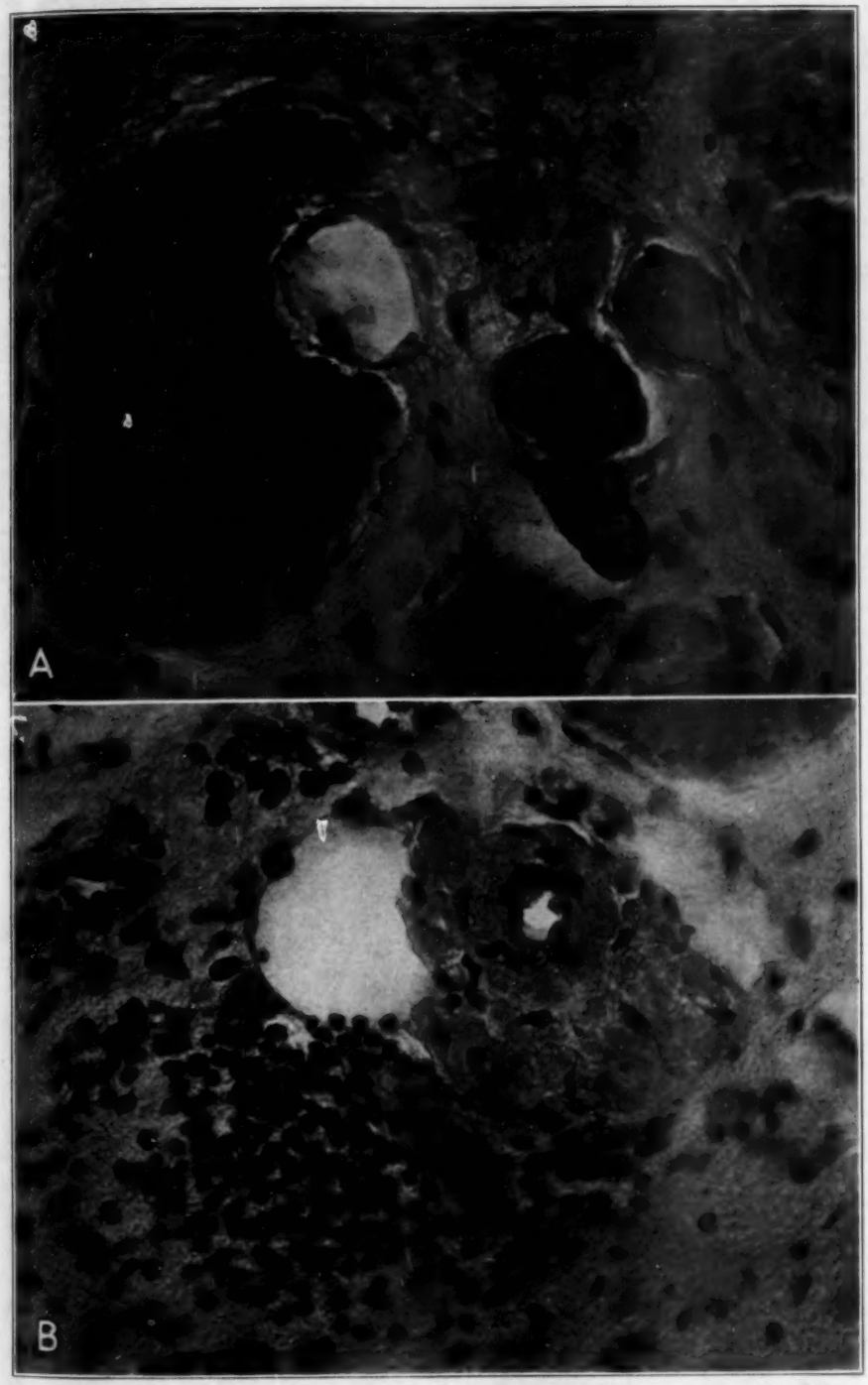

Fig. 10 (case 5).—High power detail of figure 9. *A* shows hyaline masses developing in the small blood vessels. The larger one gave an amyloid reaction. *B* shows the process of formation of hyaline (ultimately amyloid) concretions in the walls of the capillaries and the small arterioles.

to the hospital he became moderately dyspneic for two or three days. Ten days before admission it became difficult for him to breathe, and on his entrance he was seen to be markedly dyspneic.

On indirect laryngoscopy, the larynx appeared to be injected. Attached to the left vocal cord, beginning near the anterior commissure and extending posteriorally two-thirds the distance of the anteroposterior diameter of the cord, was a dark red, edematous, pedunculated, polypoid mass, which on inspiration was retracted into the glottis, rendering the patient dyspneic. There was no palpable lymphadenopathy in the neck.

As soon as a tracheotomy was performed, the dyspnea ceased. A few days later, after two attempts to remove the mass perorally, thyrotomy was finally resorted to. The tumor mass was found to be about the size of an olive; it was attached to the inferior border of the left vocal cord by a broad pedicle.

Microscopic examination of the tissue revealed that local amyloid deposits made up the greater part of the tumor.

The patient made an uneventful recovery and was discharged ten days later. Frequent examinations afterward showed no recurrence. Some hoarseness persisted (figs. 6 to 10).

FATAL COMPLICATIONS OF OTITIS MEDIA

WITH PARTICULAR REFERENCE TO THE INTRACRANIAL LESIONS IN A SERIES OF TEN THOUSAND AUTOPSIES

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AND

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LOS ANGELES

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From the Departments of Neurology of the College of Medical Evangelists and of the University of Southern California, and from the Laboratory of Neuropathology, Los Angeles County General Hospital.

A Proposed Plan for the Study of Postmortem Material in Cases with Intracranial Complications
Dura Mater
Venous Channels
Leptomeninges
Cerebral Veins
Cerebrum
Cerebellum
Summary and Conclusions

INTRODUCTION

The problem of otitis media and its fatal complications has many ramifications. One of these, the morbid anatomy of the various resultant lesions, still yields rich rewards to the investigator. Disease, however, does not recognize artificial boundaries, and we have been led more or less unwittingly into the bailiwicks of several specialties—pediatrics, internal medicine and otology. The present contribution has, therefore, expanded from a simple review of observations at autopsy to a rather complex investigation. A discussion of the important subdivisions of the subject of necessity has been reserved for later contributions.

As a basis for this study, the protocols of 10,000 autopsies performed at the Los Angeles General Hospital were reviewed. In a previous report [1] a comparison was made of the incidence of the various lesions in this series with available statistical data. This article is concerned more particularly with the characteristics of the individual lesions, their interrelationship and their possible significance.[2]

In this series the records were taken consecutively, so far as possible, although it was necessary to reject incomplete ones, and in some instances the protocols were lost. The number of records, therefore, exceeds 10,000, although the actual count reaches this figure. The head was opened whenever there was any clinical indication of an intracranial lesion. Occasionally only the intracranial contents were examined. Fortunately this did little to depreciate the value of the records, so far as otogenous complications were concerned.

The autopsies were performed, for the most part, by resident pathologists, probably with varying degrees of interest in the problems presented. It is likely that minor and less conspicuous lesions were

1. Courville, C. B., and Nielsen, J. M.: Intracranial Complications of Otitis Media and Mastoiditis: A Statistical Study, with a Survey of Ten Thousand Necropsies, Acta oto-laryng., to be published.

2. No serious attempt was made to review the almost inexhaustible literature dealing with the various phases of otogenous intracranial inflammation, and only those available reference are cited which seemed to bear directly on the major problems presented. Other contributions, concerned with more circumscribed portions of the field, will appear from time to time, in which a more representative opinion of other observers will be given.

overlooked for want of a critical examination of all the anatomic structures which might have been involved. Dr. Newton Evans, pathologist-in-chief, and his younger associates turned over the material from the last 4,500 cases to the Laboratory of Neuropathology for critical study after preliminary examination in the morgue. It is hoped that this will aid in the solution of many of the bewildering problems which confront us. The temporal bones, which were removed at the same time, were preserved for future study by members of the department of otology.

Bacteriology.—Little is to be said in this contribution regarding bacteriology. In most cases the infecting organism was identified only by a stained smear, a method which is now recognized to be of little value, particularly in differentiating between pneumococci and streptococci. Aside from occasional mention in other connections, reference to the causative organism has been made only in short case reports. In recent months, critical cultural studies were made on a series of patients with otitis media by Dr. E. M. Butt of the department of pathology of the University of Southern California, and his reports will soon be forthcoming. Only a few of these patients died, and a consideration of the bacteriologic findings in these cases would be of little value in this connection.

PROBLEMS AND PITFALLS IN THE INTERPRETATION OF OBSERVATIONS AT AUTOPSY

It is generally recognized that statistics regarding observations at autopsy are by no means an exact expression of the incidence of a given lesion or group of lesions, even though postmortem examination were possible in 100 per cent of the fatal cases. These statistics are most nearly accurate in otogenous meningitis, since it is almost invariably fatal. The figures for encephalic abscess and thrombosis of the lateral sinus, in which the mortality varies with the clinic and the surgeon, are less accurate. Autopsy records are of comparatively little value as an expression of the incidence of extradural abscess, transient reactive meningitis or minor manifestations of encephalitis, and one must of necessity, therefore, turn to the clinical records to get an approximate idea of their frequency. Cognizance of these pitfalls invariably makes such figures more valuable in the interpretation of statistics relating to postmortem examination. However, it is even more important to recognize that the greatest value of such records lies not in the incidence of lesions but in the lesions themselves and the lessons they have to teach. They may give a clue as to the mode of development of the lesions, as well as suggestions for suitable therapy.

The intracranial lesion or lesions disclosed at the autopsy table are the cause of death in most instances. At times, however, some other,

distant cause is responsible. It is in such cases, when careful study is made, that intermediate and transitional stages of intracranial lesions may be observed. This is particularly true for mural thrombi in the lateral sinuses and for manifestations of early encephalitis and minor degrees of meningeal irritation. In this country, in particular, this source of pathologic material has been largely neglected.[3]

A review of some of the problems presented by observations at autopsy is in order. The determination of the pathways of infection is now recognized to be largely a histologic problem, and mere figures or observations on gross material cannot go far in the solution of the question. One major difficulty frequently encountered is the differentiation between coincidence and consequence in a given group of lesions. At times the pathologic picture is so complex as to be hopelessly bewildering, as the following case illustrates. The variety of lesions disclosed was so great that we have come to describe it as our "catalog case."

CASE 1.—A white man, aged 44, was admitted to the hospital with the complaint of swelling of the eyes and the upper part of the nose for the previous week. Symptoms of meningeal irritation, discovered by a neurologic consultant, led to a lumbar puncture, and a turbid fluid under pressure was recovered, which was found to contain staphylococci. An antemortem blood culture demonstrated the presence of Staphylococcus aureus. The patient died six days after admission. Autopsy revealed: generalized but predominantly right-sided leptomeningitis; an acute abscess of the right temporal lobe; a bilateral subdural abscess occupying the middle fossae; bilateral involvement of the gasserian sheaths; a perihypophyseal abscess, with infectious necrosis of the hypophysis, and a pericarotid abscess on the right side. In addition, the right middle ear was filled with pus in which the ossicles were found to be floating; there was extensive necrosis of the right mastoid cells; the membrana tympani was intact; both sphenoid sinuses were filled with pus; a polypus was found in the left anterior ethmoid cells, and there was a bilateral orbital abscess, which was more extensive on the right. Furthermore, the ophthalmic veins, both cavernous sinuses, both superior and inferior petrosal sinuses and both lateral sinuses were completely thrombosed, the thrombus extending through the jugular bulb into the upper portions of the jugular veins. Figure 1 illustrates the pathologic changes. Smears and cultures of the pus from the various intracranial lesions revealed Staphylococcus aureus as the infecting organism.

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3. The modern trend to make pathology a science in itself rather than a means to an end has resulted in a profound and irreparable loss to the clinician. The clinically trained observer with little or no foundation in pathology may even find it somewhat difficult to grasp the essential details of a well written and complete report of observations at autopsy. An intelligent understanding of the pathologic processes during life is best obtained by a careful analysis of the course of the disease by the clinician with the autopsy specimen before him. A return to the clinicopathologic method, as introduced by Bonet and Morgagni more than a century ago, is greatly needed.

This unusual case has been referred to elsewhere by Love [4] to stress the possible extent and variety of pathologic alterations with an intact drum membrane. We believe that its further emphasis in this connection is warranted in order to give a complete description of the intracranial situation. Fortunately, such examples are rare.

This case illustrates not only the complexity of the pathologic picture but also the difficulty in determining which condition is the source of an

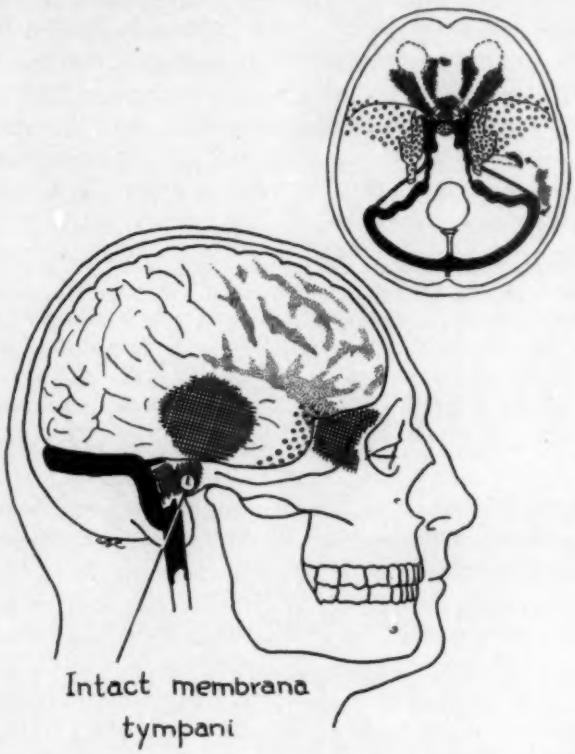

Fig. 1 (case 1).—The nature and extent of the inflammatory lesions associated with otitis media. The light stippling indicates leptomeningitis; the heavy stippling, pus in sheaths of gasserian ganglions and about hypophysis; the rings, subdural abscess; the cross-hatching, abscess of both orbits and of the right temporal lobe; the diagonal lines, purulent infection of the right middle ear and mastoid cells and of the sphenoid and left anterior ethmoid sinuses, and the solid black, thrombosis of the venous channels.

intracranial complication when both rhinologic and otologic foci of suppuration exist. This difficulty is presented particularly in a group of cases in which sphenoiditis or ethmoiditis, together with otitis media,

4. Love, A. A.: Complications of Otitis Media without Rupture of the Membrana Tympani, Arch. Otolaryng. **17**:297 (March) 1933.

is found associated with leptomeningitis. This association was emphasized by Vlasto [5] in the report of a case in which unsuspected sphenoiditis was the cause of septic leptomeningitis. He suggested that this diagnosis be considered when otitis which has been properly treated is followed by meningitis. This difficulty was also stressed by Hofer [6] in a similar case report. In a more recent consideration of the question, Eagleton [7] cited the cases of Fremel and Kramer and concluded that in such instances the meningitis is due to the infection of the sphenoid sinus. We do not know which is the responsible focus in these cases or how this can be determined with certainty in the absence of any obvious pathway of invasion.

Still another difficulty is the proper evaluation of the situation when there are two or more coincidental intracranial lesions which may or may not have consequential relationship to each other. As an example, the following case may be cited:

CASE 2.—A Mexican, aged 37, died of meningitis apparently due to acute otitis media on the right side. At autopsy erosion of the right tegmen tympani was found to be associated with a fistulous opening in the dura. An abscess of the right parietal lobe was also present, which was evidently due to retrograde extension of an infective thrombus from the right lateral sinus, which was completely occluded. Leptomeningitis was also present, the exudate being distributed particularly over the right cerebral hemisphere. The offending organism was a pneumococcus.

The question arises as to which condition was responsible for the meningitis. While it seems likely that the latter may have arisen in consequence of the direct communication between the middle ear and the intracranial space by way of the dural fistula, it could readily have been caused by an extension from the abscess of the parietal lobe or from an infected thrombus in the inferior cerebral veins or in the lateral sinus. It seems to us that the only possible solution of the question as to the sequence of events lies in an attempt to determine the age of each lesion present and to establish its position in the development of the clinical and the pathologic picture.

Fortunately, the observations at autopsy do not always disclose new problems; on the contrary, at times they clear up difficulties which have developed during the clinical course of the disease. This is espe-

5. Vlasto, M.: Meningitis of Sphenoid Sinus Origin, J. Laryng. & Otol. **42**:746, 1927.

6. Hofer, J.: Monatschr. f. Ohrenh. **60**:80, 1926; cited by Eagleton, W. P.: Intradural Complications of Aural and Nasal Origin, Arch. Otolaryng. **4**:72 (July) 1926.

7. Eagleton, W. P.: Suppurative Meningitis of Otitic and Nasal Origin: Its Relation to Blood Stream Invasion of the Pial Vessels, Arch. Otolaryng. **15**:889 (June) 1932.

cially true in the case of young children in whom signs of meningeal irritation develop during the course of acute otitis media. The natural assumption is that the infection in the middle ear has spread to the meninges, with the production of reactive or infective meningitis. When a study of the spinal fluid reveals a slightly increased cell count, with lymphocytes predominating, one may conclude that the meningeal process is as yet reactionary and give a favorable prognosis. Much to our chagrin, such findings at times proved to be signs of the early stage of frank tuberculous meningitis. The association of otitis media and tuberculous meningitis was mentioned by Portmann and Despons.[8] Further mention of this occurrence will be made in the following section, in which our own experiences are recorded.

A proper perspective of the influence of otitis media as a death-producing lesion has compelled us to view the problem in its broader aspects. In reviewing the records of this series of cases we soon realized that many patients with otitis media who came to autopsy did not have a well defined intracranial lesion but rather infectious foci elsewhere in the body or perhaps some constitutional state. In studying this phase of the subject, we reviewed the autopsy records of the last 5,000 cases. During the interval in which these were compiled one of us was associated with the department of pathology closely enough to evaluate the pathologic changes. In the following section we have considered otitis media as a fatal condition or have found its association with other conditions of sufficient consequence to produce death, aside from grossly visible intracranial extension. To this extent we have invaded the field of pediatrics.

CAUSES OF DEATH IN CASES OF OTITIS MEDIA AND MASTOIDITIS

As a terminal lesion, otitis media has been found to play a variable rôle in bringing about death. In infants it is often discovered as a terminal lesion in instances of malnutrition and prematurity in which lowered resistance to infection has probably existed. The mucous membranes of the respiratory passages, which under ordinary circumstances are more readily attacked in the early years of life, sustain the brunt of the attack of an opportunist group of organisms already resident there. In such instances a fatal issue is the result of a constitutional or a systemic disease rather than of otitis media. At other times well developed otitis media is associated with bronchopneumonia, acute gastroenteritis, an acute exanthem or, rarely, nephritis or some other and apparently unrelated condition. Here a serious question arises as to the quantitative influence of the infection of the ear in producing death.

8. Portmann, G., and Despons, J.: Méningite tuberculeuse et suppurations auriculaires ou sinusiennes, Rev. de laryng. **48**:377, 1927.

In most instances the severity of the associated inflammatory lesion is sufficient to explain the patient's death, otitis media probably being a contributing factor. It is of interest to note that even in this group of cases the brain may show some ill effects either of the local infection of the middle ear or of the attendant toxemia or both. Not infrequently pial hyperemia exists, together with an increase in the amount of fluid in the subarachnoid space ("wet brain"). It may well be argued that this reaction is dependent on the bronchopneumonia and its constitutional effect rather than on the focal infection in the middle ear.

Of the last 5,000 autopsies, which formed the basis for this more limited study, otitis media was found in 303. Of this group, 94 patients, or 31 per cent, had an intracranial infection of sufficient gravity to cause death. The remaining 326 were, for the most part, infants less than 1 year old. The highest death rate occurred during the first month of life, in which one fifth of all the deaths in the first year were found. There was then a rapid decline in the death rate up to about the sixth month, after which the rate remained almost constant (about 4 per cent a month). Of the 326 patients, only 14 were over 5 years of age. In 85 per cent of all the patients who were 2 years of age or under, both ears were affected. In a similar study of 100 cases in infants and children under 6 years of age in which section was done, Aschoff and Fuld[9] found gross evidence of otitis media in 72. Both ears were affected in 82.2 per cent of this group. The highest death rate occurred between the second and the sixth months. These figures agree closely with those of our series.

The association of otitis media with other lesions of serious import was made evident by this group of cases. In 90 some pulmonary infection (usually bronchopneumonia) was the only other discoverable lesion, and in 60 others additional lesions were found elsewhere in the body. Severe gastro-enteritis was present in 16 cases, in 4 of which it proved to be due to Bacillus dysenteriae. In 10 of this group of 16 cases an inflammation of the gastro-intestinal tract was the only other lesion present. Malnutrition as the only other cause of death beside otitis media was found in but 10 cases of this series. This condition was so evident, however, in 74 additional cases that mention was made of it in the protocol. Acute concomitant infectious disease was present in 21 cases—diphtheria in 5, pertussis in 9, erysipelas in 4 and measles in 3. It is highly probable that otitis media followed an acute infection in many other cases, but only after an interval so long that evidences of the acute disease were not evident at autopsy and not recent enough to merit mention in the protocol. Association with tuberculosis occurred

9. Aschoff, L., and Fuld, J.: Zur Frage der Otitis media bei Säuglingen und kleinen Kindern, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **23**:379, 1926.

in 15 cases; with general miliary tuberculosis in 6, with pulmonary miliary tuberculosis in 5, with glandular tuberculosis in 2 and with tuberculous meningitis in 2.[10]

It seems evident, therefore, that otitis media per se is not a lesion of serious enough consequence to cause death, unless it is accompanied by other lesions or abnormal states. It is often one of two or more contributing factors. Bronchopneumonia or some other pulmonary or pleural disease was the most common concomitant lesion in the cases studied. Other solitary associated conditions, which were less common, were acute infections, tuberculosis, gastro-enteritis and malnutrition, in that order of frequency. Infection of the respiratory passages and

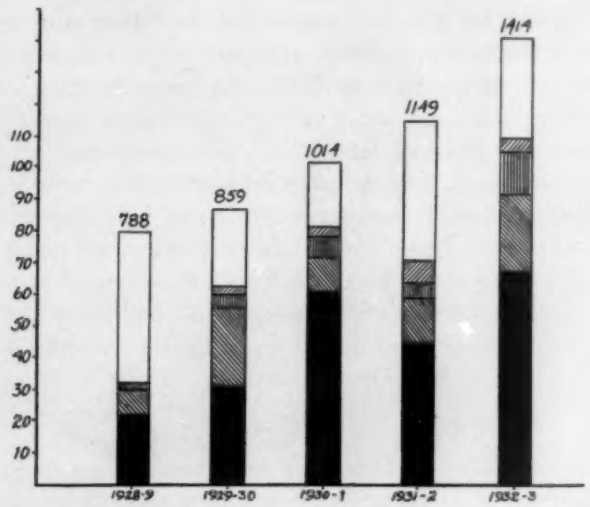

Fig. 2.—Chart showing causes of death in cases of otitis media studied during the past five years. The figures surmounting the white column indicate the number of autopsies for each year. Fluctuation in the proportion of deaths resulting from the various lesions is shown. The sections of diagonal lines running from right to left represent abscess of the brain; those of vertical lines, thrombosis of the venous channel; those of diagonal lines running from left to right, leptomeningitis, and those of solid black, otitis media with complications other than intracranial.

malnutrition were preponderantly the associated conditions of greatest significance and are probably of considerable importance in the etiology of otitis media.

10. The essential points brought out in this discussion are not new. The literature of the recent past contains numerous contributions dealing with this phase of the general problem of otitis media. Particularly noteworthy is the monograph of Grünberg (Die Erkrankungen des Gehörorgans im Kindesalter, in Brüning, H., and Schwalbe, E.: Handbuch der allgemeinen Pathologie und der pathologischen Anatomie des Kindesalters, Munich, J. F. Bergmann, 1921).

Otitis media as one of the lesions found at the time of death has been discussed in its relationship to other somatic lesions, up to this point. After the first year of life the fatalities in which this lesion plays an important rôle are caused by primary septicemia or an intracranial complication.

In most cases of septicemia or pyemia the significant lesion is thrombophlebitis of a lateral sinus, which favors the dissemination of pyogenic organisms in the blood stream. There are cases, however, in which no evidence of an involvement of the wall or lumen of the sinus is found and in which the infection has presumably gained entrance into the circulation through one of the smaller veins in the vicinity of the middle ear. Such instances of so-called primary otogenous septicemia are rare, and cases should be critically analyzed before they are so classified. Four cases in the present series of autopsies seem to fit into this clinical group, although others have probably not been detected for want of blood cultures.

The remaining group of fatal cases, those due to an associated intracranial complication, are of primary interest in this contribution. The various lesions resulting from an extension of infection of the middle ear and mastoid will be considered in anatomic order; those affecting the inner table and dura will be dealt with first, followed by a consideration of the venous channels, the leptomeninges and, finally, of the brain itself.

LESIONS OF THE TEMPORAL BONE, WITH SPECIAL REFERENCE TO INFECTION IN THE PETROUS PYRAMID

It is not our desire to discuss in detail the changes in the temporal bone in this series of cases, although certain phases of the question must be considered for the sake of completeness. Risking the possible criticism of trespassing on our neighbors' territory, we have chosen to consider certain limited aspects of this problem, since it is not likely that this series of cases will soon be reviewed in this regard. Otherwise the material dealt with here would be of considerably less value to those who are interested primarily in original infection within the temporal bone. We have limited our discussion to the cases which showed erosion of the tegmen tympani, an important step in the development of an intracranial complication, and to a consideration of so-called petrositis, the symptomatology of which is due largely to involvement of intracranial structures. In many of the cases reviewed, surgical removal of the dural and sinus plates had been effected as a part of the exploration of the mastoid, and these have not been considered. In other cases unsuspected purulent mastoiditis was found—the "masked mastoiditis" of the recent literature.

Essential Factors in Bony Necrosis.—The process of bony necrosis of the dural or sinus plates or of the bony laminae or cells of the os petrosa is considered to be infectious necrosis in which a variable and complementary rôle is played by deprivation of the blood supply on the one hand and by invasion by pathogenic organisms on the other.[11] A review of the cases of this series seems to confirm the general impression that erosion of the tegmen tympani is an indication of an essentially chronic lesion. The data obtained are not dependable enough to quote, for in many instances the occurrence of frank pus was the basis for designating a lesion as "acute," in spite of long-standing otorrhea or of repeated attacks of otitis. To obtain any degree of certainty in this respect, a review of the clinical histories in this large group of cases would have been necessary, an almost endless task.

In this series the degree and extent of necrosis of the tegmen tympani varied grossly, from softening sufficient to permit easy penetration with an instrument to a state in which a fairly large irregular opening of a diameter as great as 0.5 cm. was present. Necrosis of the sinus plate was often found to be much more extensive, especially when it was secondary to empyema of the mastoid cavity. The bony change sometimes consisted of granular disintegration (molecular necrosis) of the involved bone or, at other times, of local discoloration, with the formation of pus. When acute infection played an important rôle the characteristic reddish-green coloration of the bone and overlying dura was found (pachymeningitis), often accompanied by visible pus. In some of the cases, however, pus was found between the dura and the bone, with no grossly visible changes in the bone, a possibility already well known to otologists.

Erosion of the inner plate of the os temporale was described in 35 cases. In 27 of these the dural plate was involved, and in 18 cases of this subgroup leptomeningitis was present. In 11 of these 18 cases no other intracranial lesion was present. It is of interest that in 2 cases of thrombosis of the lateral sinus, 1 case of cerebellar abscess and 1 case of abscess of the parietal lobe, the dural plate was eroded. The possible far-reaching effects of a false interpretation based on this situation are obvious, should the otologic surgeon assume that a lesion of the temporal lobe exists when symptoms of intracranial extension have occurred simply because the dural plate is found to be eroded at the time of operation. A corollary to the rule that erosion of the dural plate is frequently followed by infection of the temporal lobe is that a coexistent lesion may be responsible for the manifestations of intracranial suppuration.

11. The matter of the nature of bony necrosis and the steps in its development have been considered in a very able manner in a recent monograph by J. Piquet (Les abcès cérébraux et leur traitement, Paris, Masson & Cie, 1913, p. 40).

In 6 cases the erosion extended to the posterior fossa [12] and in 2 it was located at the petrous apex.

Infection in the Petrous Pyramid.—When, because of the arrangement and type of the petrous cells, infection spreads medially from the middle ear into the petrous portion of the temporal bone, the condition is somewhat loosely designated as petrositis. This term is usually employed to indicate infection in the tip of the petrous bone rather than in the pars petrosa as a whole or in any other part of it. The condition is of interest to the student of neurology because its manifestations are due almost entirely to involvement of the cranial nerves crossing this region.[13]

The intracranial complications of labyrinthitis are of considerable interest. To discuss the subject intelligently it would be necessary to have a first-hand knowledge of the histopathology of this region; this is primarily an otologic problem and is therefore out of place here. In the cases considered, pus in the labyrinth was described by the more critical observers but evidently so infrequently as to render the figures of no comparative value. Two cases with bony erosion in an unusual location were studied at the autopsy table. In the first case there was extensive erosion of the midportion of the petrous ridge, just posterior to the labyrinth, the infection having had its origin apparently in the perilabyrinthine cells. In the second case the erosion extended from this situation laterally, well into the petrous angle and into a large suppurating cavity in the mastoid portion of the temporal bone. In both cases the erosion occurred on the right side. It is probable that some of the cases of meningitis and at least one case of cerebellar abscess had their origin in bony necrosis in this region. No instance of empyema of the saccus endolymphaticus was described.

Intracranial complications secondary to infection in the cells about the carotid canal are rare and are usually a matter of postmortem

12. The description given in the cases in which erosion extended to the posterior fossa were too incomplete for us to be certain as to the site and size of the eroded area. It is highly probable that many cases of focal erosion of the sinus plate and perhaps even of the dural plate have been overlooked for want of a critical examination of these regions. Small erosions at the genu of the lateral sinus are particularly apt to be overlooked by the casual observer, because this vessel so often deeply grooves the bone in this situation.

13. It is not within our province to consider the routes of infection in cases of infection within the petrous bone, a matter which has been so ably considered by Profant [18] and Kopetzky and Almour.[20] In this connection it is more essential to know that infection may be localized (*a*) in the perilabyrinthine cells (perilabyrinthitis), (*b*) in the cells about the carotid canal (pericarotiditis petrosa) or (*c*) in the cells of the petrous apex (apicitis petrosa), or may not be localized but (*d*) may involve the entire central portion of the petrous bone (empyema of the petrous bone, osteomyelitis of the petrous bone, petrositis universalis).

discovery. There may be erosion of the carotid canal, with the characteristic discoloration or molecular necrosis of the bone in this region. A pericarotid abscess may result, at times associated with involvement of the arterial wall (arteritis) or thrombosis of the pericarotid network of small veins. Should partial or complete thrombosis of the artery occur, focal or general infarction of the brain may result, rarely with the formation of an abscess.[14]

A group of several cases in which there was involvement of the bone forming the carotid canal and of the artery itself was disclosed in this review. They are of sufficient rarity to merit a brief description of the observations.

CASE 3.—Autopsy was performed on a Mexican, aged 30, who died following an acute attack of right-sided otitis media and mastoiditis. Extensive thrombosis of the entire intracranial portion of the right internal carotid artery, the right ophthalmic artery and the basilar and right vertebral arteries was found. While not specifically mentioned, it is likely that the right posterior communicating artery, an important link in this chain of vessels, was also thrombosed. The brain substance of the right cerebral hemisphere was rather pale, but no areas of softening or of abscess formation were described. The patient died of septicemia. The infecting organism was a hemolytic streptococcus.

CASE 4.—A boy of Mexican parentage, aged 2 years, died following an acute attack of otitis media bilateralis associated with bronchopneumonia. Postmortem examination revealed that the intracranial portions of both internal carotid arteries were almost completely occluded with adherent antemortem thrombi, the local lesion being verified microscopically. While the brain tissue as a whole was somewhat softer than usual, no focal areas of necrosis or abscess formation were disclosed. Pneumococci were isolated from the pus in the middle ears.

CASE 5.—A white man, aged 52, died with symptoms of meningitis after a long history of recurring attacks of otorrhea on the left side. Necropsy revealed apicitis petrosa on the left side, which was characterized by the occurrence of reddish-green discoloration of the dura and bone in this region. There was a collection of pus about the left carotid artery in its bony canal. When the artery was dissected out it was found to be discolored and ragged, due to invasion of its adventitia. The lumen, however, was entirely free from any evidence of thrombus formation. Basilar meningitis was coexistent, due to Haemophilus influenzae.

Two other examples of pericarotid abscess were found in this series of cases. One of these, the etiology of which was uncertain, although it was associated with homolateral disease of the middle ear, has already been discussed. The abscess about the carotid artery was one of the many lesions in our catalog case (case 1). In the other case, erosion of the bone forming the carotid canal and a pericarotid abscess were consequent to extensive osteomyelitis of the petrous bone. This case will

14. Körner (Die otitischen Erkrankungen des Hirns, der Hirnhäute und der Blutleiter, ed. 5, 1902, Munich, J. F. Bergmann, p. 207) refers to two cases of cerebral embolism following otogenous thrombosis of the carotid artery, those of Gairdner and Barr and of Baizeau.

be discussed subsequently (case 10). No doubt other cases in which there were minor changes in the bone about the carotid artery escaped attention. Since this series was closed such a case came to light. Focal necrosis of bone was found in this region in a patient who had persistently complained of deep-seated pain in the temporal region.

Apicitis Petrosa.—The location of infection in the tip of the petrous bone is of more common occurrence. Stimulated by Gradenigo's original contribution, many investigators have reported cases presenting the characteristic syndrome which he described, and much has been written concerning the pathways of infection and the mechanism of the production of symptoms. We shall confine ourselves to a consideration of the intracranial complications of this interesting lesion. In view of the rarity of the cases in which autopsies have been performed, a short résumé of the findings in the cases in this series will be given.

CASE 6.—A white man, aged 46, who gave a long history of left-sided otitis media, died of septic meningitis. At necropsy the tip of the left petrous bone was found to be eroded, and a small local abscess was disclosed when the dura was removed. Over the dorsolateral surface of the left cerebral hemiphere a thin yellowish purulent exudate was found. The infecting organism proved to be a streptococcus.

CASE 7.—A white man, aged 27, with a history of chronic otitis media on the left side, died of pneumococcic meningitis. In addition to the thick yellowish fibrinopurulent exudate over the dorsolateral surface and at the base of the right cerebral hemisphere, a suppurating focus in the apical cells on the left was found. There were no gross local pathologic changes in the dura. The infecting organism was a pneumococcus.

CASE 8.—A Negro, aged 46, died following left-sided otitis media of six weeks' duration which terminated with the characteristic symptoms of meningitis. At autopsy the bone was found to be eroded in the region of the petrous apex, the anterosuperior surface being involved. A local extradural abscess was also found. There was generalized meningitis of pneumococcic origin. Well defined bronchopneumonia was also present.

CASE 9.—A Caucasian man, aged 41, died with symptoms of meningeal irritation following right-sided otitis media of four days' duration. At necropsy erosion of the bone at the tip of the right petrous bone was disclosed, with an associated extradural abscess. The bone forming the carotid canal was also eroded. There was generalized septic meningitis due to invasion by the pneumococcus.

A sixth case in which an infection in the tip of the petrous bone was found has already been mentioned (case 5). It was similar to case 9 in that there was erosion of the carotid canal as well as of the petrous apex. The findings in these cases indicate that infection may become localized in more than a single focus within the petrous pyramid. Had the cells about the labyrinth been studied microscopically in all the cases, no doubt foci of infection would have been discovered in more of them.

The confluence of multiple inflammatory foci in the petrous pyramid with progressive suppuration in these areas may lead to the isolation and sequestration of a central mass of bone. At times the entire central portion of the bone is hollowed out by the suppurative process. This more extensive involvement of the pyramid has been called generalized osteomyelitic petrositis or osteomyelitis of the pyramid. In line with the nomenclature of this group of lesions, the designation of petrositis universalis is suggested.

CASE 10.—A white boy, aged 12, died with symptoms of meningitis superimposed on a chronic otitis media. Autopsy disclosed extensive necrosis of the entire central portion of the right petrous bone, with the formation of a gross suppurating cavity. The carotid canal was also eroded, and a local pericarotid abscess was present. The right cavernous and superior petrosal sinuses were thrombosed. There was also advanced basilar meningitis; a pneumococcus was isolated from the exudate.

CASE 11.—A Mexican girl, aged 3 months, died five weeks after the onset of diarrhea which had been followed shortly by symptoms of pertussis. Erysipelas developed on the right cheek, along with bilateral otitis media. At autopsy the entire central portion of the left petrous bone was found to be necrosed, the resulting cavity being filled with dark, foul-smelling material. The right petrous bone was similarly but less extensively involved. A stained smear revealed staphylococci, short-chained streptococci and short gram-negative bacilli.

One interesting observation is worth mention in passing. In 3 of the cases already described (cases 5, 9 and 10) there was erosion of the bone in the carotid canal as well as in the petrous apex, and in 2 of these cases a pericarotid abscess existed. In 1 of these 2 cases labyrinthitis was also discovered. Thus infection in the petrous bone may involve one group or all groups of cells, leading to extensive osteomyelitis of the pyramid.

A review of this group of cases with infectious involvement of the petrous pyramid indicates the often complex status of this condition, the variety of its manifestations and the great frequency of septic meningitis as a part of the terminal pathologic picture. It is now in order to consider in more detail the nature of the intracranial lesions which arise consequent to otogenous suppuration of the petrous bone.

Intracranial Complications of Apicitis Petrosa.—When death occurs after clinical evidence of apicitis petrosa, some intracranial complication is likely to be present at autopsy. In rare instances the fatality may be due to associated septicemia or bronchopneumonia. Strangely enough, extensive osteomyelitis may be unaccompanied by any intracranial lesion (case 11). The complications which may occur are leptomeningitis, pachymeningitis, thrombosis of the venous channels, infection of the gasserian sheath and, possibly, abscess of the temporal lobe. An unquestioned case of cerebellar abscess secondary to infection in the petrous apex has not come to our attention.

Of all the intracranial complications of apicitis petrosa, septic leptomeningitis is the most common. In 1927, Sears [15] collected from the literature a series of 210 cases of paresis of the abducens nerve, associated with otogenous intracranial complications; there were 43 deaths, 31 of which were due to meningitis. In the series of cases of Gradenigo,[16] which were collected three years after his original description of the syndrome which bears his name, 4 of the 57 patients died of meningitis. In 1925 the same author described possible routes of infection to the meninges.[17] In our series of 7 cases of apicitis petrosa, 6 gave evidence of septic meningitis at autopsy (generalized in 4, basilar in 2). In a recent contribution, Profant [18] collected 19 cases in addition to those considered in his first paper, including 3 more of his own. In 6 of these cases death was evidently due to meningitis.

The pathways of infection to the meninges must of necessity be legion. It should be remembered that either the original focus or some secondary complication (labyrinthitis, thrombosis of a venous channel) may be the source of meningeal infection. This question was considered recently by Lawson,[19] who reported 3 additional cases which were verified by autopsy. Infection may reach the meninges through the point of erosion or the local extradural abscess thus formed, through the internal acoustic meatus or through the medium of some associated or consequential lesion. One of the cases of Lawson, 1 of Kopetzky and Almour [20] and 1 of Eagleton [21] are of interest in that there was erosion into the posterior fossa, a process which occurs more commonly on the anterior surface of the apex.

The various inflammatory lesions of the dura have been grouped under the collective term pachymeningitis. The two characteristic lesions are the local apical extradural abscess and inflammatory discoloration

15. Sears, W. H.: Otogenic Pathology in Paralysis of the Abducens Nerve, Arch. Otolaryng. **5**:462 (May) 1927.

16. Gradenigo, G.: Ueber die Paralyse des Nervus abducens bei Otitis, Arch. f. Ohrenh. **74**:149, 1907.

17. Gradenigo, G.: Rev. oto-neuro-opht. **2**:158; cited by Eagleton, W. P.: Intracranial Complications of Aural and Nasal Origin, Arch. Otolaryng. **4**:171 (Aug.) 1926.

18. Profant, H. J.: Gradenigo's Syndrome, with a Consideration of "Petrositis," Arch. Otolaryng. **13**:347 (March) 1931. This author apparently does not include Sears's cases in his collection.

19. Lawson, Lawrence J.: The Problem of Meningeal Infection from Petrositis: Report of Cases, Arch. Otolaryng. **17**:640 (May) 1933.

20. Kopetzky, S. J., and Almour, R.: The Suppuration of the Petrous Pyramid; Pathology, Symptomatology and Surgical Treatment: IV. Case Reports, Ann. Otol., Rhin. & Laryng. **40**:953 (Sept.) 1931.

21. Eagleton, W. P.: Localized Bulbar Cisterna (Pontile) Meningitis, Facial Pain and Sixth Nerve Paralysis and Their Relation to Caries of the Petrous Apex, Arch. Surg. **20**:402 (March) 1930.

(pachymeningitis interna). Extradural abscess was found in 3 of our 5 cases, always with a local bony lesion. Erosion without abscess but with local pachymeningitis occurred in 1 case, and neither erosion nor abscess occurred in 1. Extradural abscess is a rather common finding at autopsy in apicitis petrosa, according to the most recent observers. Kopetzky and Almour [20] referred to the cases of Uffenorde, Ostmann and Grunert, in which there were extradural abscesses secondary to this condition, while in the case described by von Troeltsch there were two such abscesses, a rather unusual finding. The pathologic characteristics of pachymeningitis interna (pachymeningitis penetrans) are to be described in a later section.

Thrombosis of a venous channel resulting from apicitis must be rare. In the few reported cases of cavernous thrombosis associated with infection in the cells of the tip, direct communication or consequential connection was not established.[22] The possibility of extension directly from an infected apical cell was admitted by Eagleton.[23] He cited the cases of Ballance, Blake and Charlin as instances. The last one is not a clearcut example, because an orbital abscess, which is the cause of an infection of the cavernous sinus in some instances, was also present. The author described 1 case which had come under his personal observation, but this likewise was not entirely typical, for the infected cells were located particularly about the carotid canal, and infection may well have spread to the sinus by way of the pericarotid venous plexus.

In Brown's [24] case a patient presenting Gradenigo's syndrome died with thrombosis of both cavernous sinuses. Unfortunately the petrous bone was not removed, nor were the petrosal sinuses critically studied. The case is therefore open to question. In 1 of our cases homolateral thrombosis of the cavernous sinus and the superior petrosal sinus was found, associated with petrositis universalis. It seems likely that the thrombus in the cavernous sinus was secondary to that in the superior petrosal sinus. A most unusual case of thrombosis of an inferior petrosal sinus secondary to apicitis petrosa (with an extradural abscess) was reported by Atkins.[25]

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22. This is of interest, particularly as some of the venous channels from the middle ear communicate with the cavernous sinus and the inferior petrosal sinus, as Pietrantoni (Arch. ital. di otol. **38**:296, 1927) seems to have shown in specimens in which injections were made.

23. Eagleton, W. P.: Cavernous Sinus Thrombophlebitis and Allied Septic and Traumatic Lesions of the Basal Venous Sinuses, New York, The Macmillan Company, 1926, p. 36.

24. Brown, L. G.: Gradenigo's Syndrome: Mastoidectomy Followed by Bilateral Cavernous Sinus Thrombosis, J. Laryng. & Otol. **44**:601 (Sept.) 1929.

25. Atkins, R. Travis: Suppuration of the Petrous Pyramid; Rupture of Extradural Abscess Over the Apex; Suppurative Cerebrospinal Meningitis; Death; Autopsy, Laryngoscope **38**:404, 1928.

It seems most unlikely that abscesses within the temporal lobe are ever the direct consequence of infections in the tip of the petrous bone, although in such cases symptoms of petrositis have occasionally been recorded. For example, Sears [26] collected 6 cases of abscess of the temporal lobe in his review of the literature dealing with otitic paralysis of the nervus abducens. In the 2 cases which were verified by autopsy, no mention of infection in the tip of the petrous bone was made, and, furthermore, local adhesion to the tegmen tympani gave every indication that the abscess was the result of direct extension through the dural plate. The same condition seemed to be present in the case reported by Roberts.[27] Eagleton [28] found but 1 such case in his extensive practice.

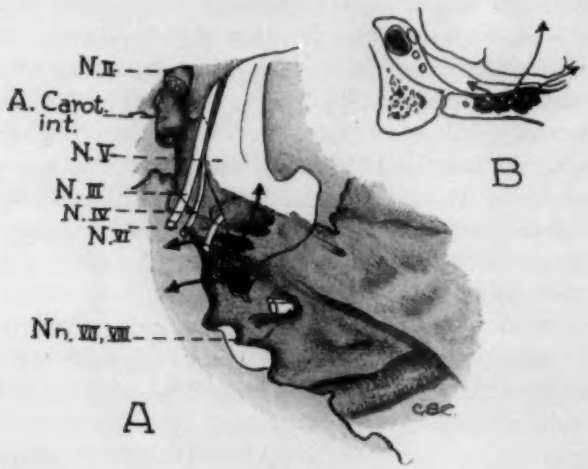

Fig. 3.—Drawings showing the mode of extension into the intracranial space consequent to infections of the petrous apex: *A*, erosion of bone in either the middle or the posterior fossa, with extension into the subarachnoid space or into the sheaths of adjacent nerves (as the trigeminus); *B*, cross-section of the tip of the petrous bone, showing extension into adjacent spaces and structures. A theoretical but questionable extension into the temporal lobe to cause abscess is also indicated.

There was no evidence of petrositis in any of the cases of abscess of the temporal lobe in this series which came to autopsy.[29]

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26. Sears, W. H.: Otogenic Paralysis of the Abducens, with Especial Mention of Isolated Palsy Associated with Irritation of the Gasserian Ganglion, Tr. Am. Laryng., Rhin. & Otol. Soc., 1925, p. 89.

27. Roberts, E. R.: Acute Otitic Temporosphenoidal Abscess, Arch. Otolaryng. **6**:420 (Nov.) 1927.

28. Eagleton, W. P.: Personal communication to the authors.

29. We had the opportunity to study a case clinically in which rather typical manifestations of apicitis petrosa were found. The patient died of septic meningitis which was apparently due to rupture of an abscess of the temporal lobe. Unfortunately, permission for autopsy could not be obtained.

Subdural abscess secondary to infections in the petrous apex is evidently rare. Roberts [27] found pus in the middle fossa in his case, associated with an abscess of the temporal lobe. Leutert's [30] patient had a local subdural abscess in the region of the gasserian ganglion. It would be necessary to rule out all possible causes of subdural abscess, such as thrombosis of the cavernous sinus, orbital abscess, purulent sphenoiditis and other conditions, in order to prove that the abscess was secondary to apicitis. Although such a case was not observed in our series, the possibility must be admitted.

INFECTION OF THE DURA MATER, INCLUDING EXTRADURAL AND SUBDURAL ABSCESSES

Otogenous infections of the dura mater necessarily include a wide variety of lesions, microscopic as well as macroscopic. It is obvious that minute lesions could easily be overlooked in any study of gross intracranial lesions. Furthermore, many dural lesions are essentially surgical, clearing up spontaneously after proper operative treatment. This is especially true of local deposits of fibrin, granulation tissue, or small epitympanic extradural abscesses. Even when such a lesion has existed prior to death, macroscopic evidence of its presence may be wanting at autopsy. It is evident at the outset that the figures given here as to the incidence of dural infections cannot be taken as representing the true situation. In this group of lesions, extradural abscess, local pachymeningitis (penetrans), dural scarring, subdural abscess and dural fistula are to be described.

Extradural Abscess.—The term extradural abscess as used in the literature is often indefinite, either as to the amount of localized pus or as to its location. We have designated as extradural abscess any grossly visible collection of purulent exudate between the skull and the dura. It seems advisable to describe these collections in such a way as to indicate their location, as is already the custom in designating perisinuous or epitympanic abscess. Such extradural collections of pus may also occur along the ridge or at the apex of the petrous bone. The latter two lesions are essentially postmortem conditions, and, when found, should be described accurately enough for the reader to know just where the pus was situated. It is theoretically possible to have an extradural collection of pus secondary to infection in the zygomatic cells or in the squamous portion of the temporal bone. The occurrence of pus outside the dura is usually an indication of a fairly high degree of local resistance, which accounts for the fact that it is found so many times at operation with no other intracranial lesion.

30. Leutert, E.: Bakteriologische Untersuchungen der akuten Warzenfortsatzempyem mit besonderer Berücksichtigung der Pneumokokkeninfektion des Ohres, Verhandl. d. deutsch. otol. Gesellsch., 1896, p. 62.

In this series of autopsies a thickened dura in the middle fossa was found twice; in each instance it was associated with a subdural abscess. In 2 other cases the dura was said to be adherent to the temporal bone, due to old local adhesions. Had microscopic sections been made as a routine procedure, it is likely that an interesting array of lesions would have been disclosed.

Extradural abscess was found about the lateral sinus in 6 cases, at the tip of the petrous bone in 3 and above the tegmen tympani in 10—19 cases in all. In the group last mentioned, 2 instances were noted in which a very small extradural abscess was associated with an abscess of the temporal lobe. In another case an extradural abscess was associated with a cerebellar abscess (with no apparent connection); in 1 case it was associated with a subdural abscess and in 6 cases with meningitis. In 4 of the cases meningitis was the only intradural condition, although it may not have been consequent to the extradural abscess. In 1 case an epitympanic abscess existed, with no gross evidence of underlying bony change, and in 2 others small abscesses were present, in spite of surgical removal of the tegmen tympani. They were located somewhat farther forward over the tympanum.

Pachymeningitis.—In this discussion we are not concerned with pachymeningitis externa, which has been considered along with extradural abscess. The lesion here considered should be described as pachymeningitis penetrans rather than pachymeningitis interna, since both layers of the dura are affected. This condition is evidently the result of a fairly rapid, progressive infection, indicated in our cases by discoloration and softening of the underlying bone. The gross appearance of the lesion was characteristic, regardless of its situation. There was a small round or oval patch of gelatinous or stringy exudate, and the dura was a mottled reddish yellow-green. When located over the tegmen tympani or the tip of the petrous bone, the lesion was apt to be circumscribed, but it might be more extensive over the lateral sinus. In this situation the bone forming the groove of the sinus might have a more greenish coloration, due possibly to breaking down of blood pigment. This type of lesion was found in 6 cases, in 5 of which it was situated over the tegmen tympani and in 1 over the tip of the petrous bone. In all 6 cases the lesion was associated with gross bony erosion and discoloration, and in each case leptomeningitis was present, which suggests the virulent nature of the small amount of subdural exudate.

Subdural Abscess.—The subject of otogenous subdural abscess seems to have received scant attention, if its treatment in the available literature is any indication. There are, no doubt, definite reasons why a subdural abscess is overlooked, among which might be mentioned its association with other and more striking lesions and the tendency to

confuse it with leptomeningitis. The comparative rarity of subdural abscess secondary to suppuration of the middle ear stands out in marked contrast to its frequency consequent to infection of the frontal, ethmoid or sphenoid sinuses.

In the present series, 17 cases of subdural abscess were associated with otitis media. In case 1 the abscess was very likely due to the bilateral orbital infection or the purulent sphenoiditis, and in another case it may well have been caused by associated sphenoiditis. Fifteen cases remain, therefore, in which a subdural abscess seemed to be due directly or indirectly to infection of the middle ear. From this group of cases a classification of the etiology of the condition has been elaborated.

Classification of Subdural Abscesses on an Etiologic Basis

A. Extension by direct continuity through the bone and dura:

1. Direct spread, with no local reaction from the point of penetration through the bone and dura, usually over the tegmen tympani (3 cases)
2. With active local reaction and walling off of a small local subdural abscess, usually located between the posterior surface of the petrous bone and the anterior margin of the cerebellum (4 cases)
3. Secondary breaking down of adhesions about a dural fistula, leading to a superficial cortical ulceration (1 case) or to an abscess in the temporal lobe (1 case)

B. By retrograde extension along venous channels:

1. Regional, about the inferior cerebral veins or (rarely) about the superior cerebellar veins, afferent channels of the lateral sinus which was thrombosed (3 cases)
2. Distant, consequent to extension to distant points of the thrombotic process originating in the lateral sinus.
   (*a*) Extension to the cavernous sinus (1 case) or to the superior longitudinal sinus (1 case)
   (*b*) Extension by way of the anastomotic venous system (1 case)

C. Rupture of an abscess of the brain into the subdural space:

1. Spontaneous rupture[31] (0 cases)
2. Postoperative rupture into the subdural space following attempts at drainage (1 case—posterior fossa)

Mygind's[32] report is unique in the high proportion of subdural abscesses. In a series of 207 complications of otitis media, he found

31. The spontaneous rupture of a large abscess of the brain into the subdural space may be considered a possible cause of subdural abscess. While no case of this type was found in the 10,000 cases included here, 1 subsequently came to our attention. An abscess of the left frontal lobe, associated with purulent sphenoiditis ruptured through the adherent leptomeninges into the subdural space, with the formation of a large abscess.

32. Mygind, H.: Otogene multiple intracranielle Krankheiten, Ztschr. f. Ohrenh. **81**:307, 1921.

19 instances of this lesion (9.2 per cent). In 1 of his cases it occurred as a solitary lesion, although an extradural abscess might also have been present, since this lesion was ignored in his computations. In 14 of these 19 cases leptomeningitis was also present. Although it was found in 10 cases of abscess of the brain, it was never the only other lesion. Sinus phlebitis was also present in 10 cases. In a more recent contribution on the subject, Rüedi [33] described 5 cases of subdural abscess,

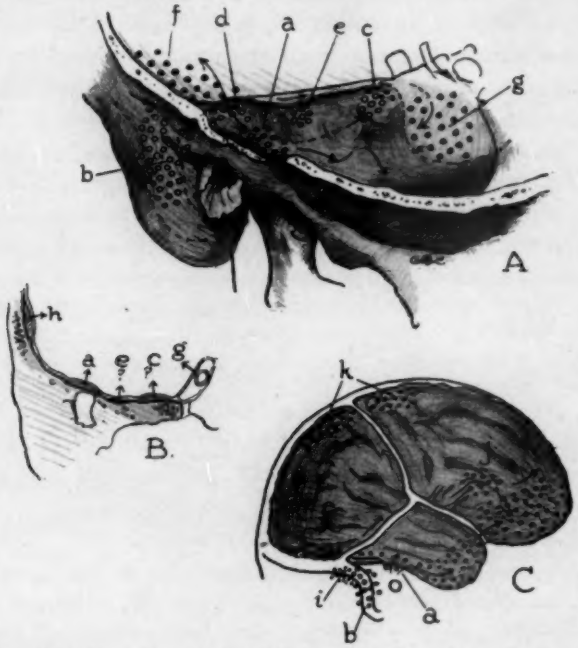

Fig. 4.—Localization of extradural (circles) and subdural (dots) collections of pus. *A* shows locations in the region of the temporal bone. *B* shows possible points of direct extension into the intracranial space. *C* shows focal and distant localization of subdural abscesses. The arrows indicate the points of invasion and routes of extension: *a*, extension through the tegman tympani and antrums; the subdural abscess may extend into the depths of the middle fossa, anteriorly over the frontal lobe or posteriorly onto the superior surface of the tentorium; *b*, perisinuous infection; *c*, extension through the tip of the petrous bone, with the theoretical possibility of a formation of subdural abscess; *d*, extradural abscess along the ridge of the petrous bone; *e*, theoretical extradural and subdural abscess secondary to labyrinthine or perilabyrinthine infection; *f*, subdural abscess secondary to thrombosis of the cavernous sinus; *g*, subdural abscess of the gasserian sheath; *h*, extension from the focus of osteomyelitis in the squamous portion of the temporal bone; *i*, subdural abscess in the region of the genu of the lateral sinus; *j*, distant subdural abscess (as frontal) secondary to thrombosis of the cerebral veins; *k*, subdural abscess along the superior longitudinal sinus.

33. Rüedi, L.: Der otogene Subduralabszess, Monatschr. f. Ohrenh. **64**:3 (Jan.) 1930.

3 of which were due to direct extension through the bone. In another case the subdural abscess was at some distance from the original infection in the ear and may have been due to retrograde extension.

Otogenous subdural abscesses are more apt to be localized than those originating from frontal sinusitis. This is particularly true of those directly consequent to extension through the tegmen tympani. In 1 of our cases, however, a rather extensive subdural abscess resulted from infection by way of the tegmen tympani. This matter will be considered more critically in a later contribution.

In our series of 17 cases (including 2 which are open to question as possibly being due to accessory nasal infection), leptomeningitis was present in 13. When a subdural abscess was associated with an extradural abscess, leptomeningitis was an invariable accompaniment. In 3 of our cases in which a subdural abscess was not associated with leptomeningitis, thrombosis of the lateral sinus was the only other lesion. It is apparent, therefore, that leptomeningitis, either directly or indirectly, through rupture of an abscess of the brain, is the cause of death in from 75 to 80 per cent of the patients, and the remainder die as the result of thrombosis of the lateral sinus. In the 15 unquestioned cases, acute otitis media was associated in 6 and chronic otitis media in 8, while 1 was uncertain. Of the 2 cases in which we were doubtful as to the otitic origin of the abscess, 1 was associated with acute otitis media and 1 with chronic otitis media. Thus it would seem that the lesion is more apt to follow chronic infections of the ear, as Liebermann and Petzal [34] suggested. A great many factors must of necessity be considered, because of the wide variation in the pathogenesis of this lesion.

Dural Fistula.—The references to dural fistula in the literature usually pay particular attention to abscesses of the temporal lobe having a so-called stalk. Mention also has been made, though rarely, of an opening in the dura leading to a cortical abscess, otherwise known as a superficial cortical ulceration. In the present series, an actual gross opening in the dura associated with an opening through the bony tegmen tympani into the middle ear or antrum was found in 7 cases. No dural fistula was found elsewhere.[35] In 2 of these cases the fistula was asso-

34. Liebermann and Petzal: Zur Pathologie der otitischen Subduraleiterung, Ztschr. f. Hals-, Nasen- u. Ohrenh. **15**:294, 1926.

35. One is caused to wonder why fistulous openings in the dura do not occur in other locations. It is probably because no other part of the petrous bone is so subject to chronic bony changes as the region of the tegmen tympani. Chronic bony changes are necessary for the production of disintegration sufficiently extensive to be indicated as fistula. On the other hand, perisinuous infections are often very extensive, and the entire sinus plate may be eroded even after a relatively acute infection. The anatomic situation seems to predetermine the type and extent of bony destruction.

ciated with a stalk leading to a large abscess in the temporal lobe; in 3 cases septic meningitis was the only intracranial lesion, and in another case a subdural abscess was associated with leptomeningitis.

The size of the dural fistula varied considerably. In some instances it was the size of a pinpoint and could not be discovered grossly. In 1 of our cases, in which the fistula was secondary to a local extradural abscess, its presence was indicated only when slight pressure over the area caused a drop of pus to appear at the site. In other cases on opening 1 cm. or more in diameter was found. In a somewhat unusual case there was a fistula in the bone and dura in the region of the right tegmen tympani, with associated thrombosis of the right lateral sinus, an abscess in the right parietal lobe and septic meningitis (case 2). In another case the fistula, the external opening of which was hidden in a mass of granulation tissue on the outer surface of the dura, led to erosion of the cortex. Rupture of the protecting layer of adhesions about the fistula resulted in a local subdural abscess and terminal meningitis. This case will be referred to later in another connection (case 15).

THROMBOSIS OF THE INTRACRANIAL VENOUS CHANNELS

Otogenous thrombophlebitis of the intracranial venous channels, commonly thought of in terms of thrombosis of the lateral sinus, presents an unusual variety of manifestations and may be attended by widespread and complex pathologic pictures. In its simpler aspects it is a contiguous infection involving the smaller radicals or the wall of the lateral sinus itself, resulting in the formation of a thrombus. A small mural thrombus may resolve spontaneously, may go on to more extensive development, with dissemination of infectious material in the blood stream, or may break down to form an abscess within the confines of the wall of the sinus. More complex conditions result from widespread retrograde extension of the thrombus or from a distant localization of infectious material by the retrograde flow of particles into the venous tributaries, favored by a dammed-up blood stream. This may lead to the establishment of infectious foci in the brain at a considerable distance from the original thrombus and without any demonstrable continuity with it. In infants, the additional factors favoring primary thrombosis—anemia, malnutrition and dehydration—may result in widespread occlusion of the venous channels draining the cranial vault, with characteristic changes in the substance of the brain.

Considering each channel separately, table 1 indicates the number of times each was involved.

Each group of lesions, with the particular pathologic characteristics, will be considered separately.

Thrombosis of the Lateral Sinus.—This condition was present in 35 cases in the present series, being unilateral in 31 and bilateral in 4. In but 3 of these was there found no other detectable associated lesion of sufficient severity to cause death. One of the 3 patients had pulmonary tuberculosis. The other 2 had a thrombus extending into the jugular vein, and undiscovered septicemia may well have existed. The presence of septicemia was established by blood culture in 4 cases, and pyemia occurred in 2 others. Extension into other venous channels occurred in 11 of these 35 cases. Septic meningitis was present in 12 and a subdural abscess in 3.

The alterations disclosed at autopsy were usually in an advanced stage, although in 1 recent case particularly careful exploration revealed

TABLE 1.—*Incidence of Otogenous Thrombosis of the Intracranial Venous Channels*

Lateral sinus*		35
Unilateral	31	
Bilateral	4	
Cavernous sinus		9
Superior longitudinal sinus		8
Superior petrosal sinus		4
Unilateral	2	
Bilateral	2	
Inferior petrosal sinus		3
Unilateral	2	
Bilateral	1	
Cerebral veins		10
External †	8	
Internal	2	
Total		69

* This includes a single case of thrombosis of the jugular bulb, which will be referred to later (case 12).

† This figure is probably too low. Retrograde extension into the inferior cerebral veins and, in particular, the cerebellar veins is not an unusual finding at autopsy. Probably no search was made for these small local extensions in all instances.

a small mural thrombus, and in 5 others the clot was found to be small, averaging 5 cm. in length and located in the sigmoid portion of the sinus. These patients died of septicemia (2 cases), of serious pulmonary infection, such as pneumonia (2 cases), or of abscess of the brain (1 case). A rather frequent finding was an extension of the clot into the upper portion of the internal jugular veins. In 24 of the 35 cases in which this was noted, the thrombus also extended to the torcular herophili, beyond it into the opposite sinus (21 cases) or within from 3 to 5 cm. of it (3 cases).

In one case primary thrombosis of a jugular bulb was found. For the sake of completeness, a short résumé of the findings in this case is included here.

CASE 12.—A white boy, aged 9 years, came to the hospital with acute otitis media and a swelling of the right side of the neck, which had developed during an attack of measles. He died five days later. At autopsy extensive erosion of

the mastoid cells on the right, with an extension of the infection into the lateral portion of the petrous bone (the extent was not stated), was found. There was primary thrombosis of the jugular bulb on the right, with a secondary extension into the internal jugular vein. This was the source of a cervical abscess which involved the superficial tissues of the neck. The infecting organisms, as found in the right middle ear and in the thrombus of the jugular bulb, were streptococci and staphylococci.

The description of the thrombus itself, of the internal and external appearance of the wall of the sinus and of the adjacent bony changes in this series was too meager to be of any benefit except in the cases which were studied personally by us.[36]

An intrasinus abscess was observed in 2 cases recently studied, which are of sufficient interest to merit a brief record here.

CASE 13.—A Caucasian youth, aged 17, complained of pain in the left shoulder, of six months' duration. This subsequently proved to be due to staphylococcic osteomyelitis of the shoulder joint, of unknown origin. Two months before death, pain was noticed in the left ear. Increasingly severe headaches, vomiting, blurring of vision, drowsiness and right homonymous hemianopia led to exploration of the left temporal lobe, with negative results. The patient died of pneumococcic meningitis. At autopsy no abscess of the brain was found, but there was extensive basilar meningitis. The left lateral sinus contained free pus. Its tributary veins were thrombosed.

CASE 14.—A Mexican boy, aged 15, who suffered from diabetes, died with symptoms of meningitis five weeks after the onset of acute otitis media and five days after ligation of the left internal jugular vein. At the time of operation, pus was found in the lateral sinus and in the jugular vein as far down as the neck could be explored. At autopsy pus was found to extend into the adjacent portion of the left innominate vein and was separated by only a thin layer of thrombus from the open blood stream. Pus mingled with a recent thrombus was also found above the pack in the lateral sinus. The infecting organism was a streptococcus.

It is possible that other instances of intrasinus abscess are included in this group of cases, but the descriptions were too incomplete for them to be recognized as such.

Metastases from the original thrombus may be the result of hematogenous infection, the release of thrombotic particles into the blood current or retrograde extension by an obstructed venous current. Metastases resulting from extension in the direction of the venous flow probably occur fairly early in the course of the disease, when the current of blood is washing over the surface of a mural thrombus. At times

36. Considerable work has been done on the pathology of lateral sinus thrombosis in recent years. Of the recent contributions on the subject, that of Haymann (in Denker, A., and Kahler, O.: Die Krankheiten des Gehörorgans, Handbuch der Hals-, Nasen- Ohrenheilkunde, Munich, J. F. Bergmann, 1927, vol. 8, p. 54) may be taken as a classic example.

they are due to breaking up of a thrombus by disintegration, even after complete obstruction has taken place. The infected particles enter the pulmonary circulation after passing through the right side of the heart. Peripheral metastases are produced by bacterial emboli or particles small enough to pass through the pulmonary filter. Figure 5 shows the location of metastatic foci, as found in this series of cases. It is readily seen that the lungs and pleurae are the structures most frequently involved.

The lesions produced by retrograde extension into contiguous sinuses or tributary veins, including subdural abscess or hemorrhage, lepto-

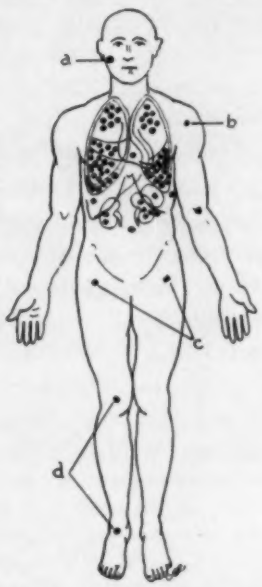

Fig. 5.—Locations of metastatic foci in patients dying of thrombosis of the lateral sinus: *a*, abscess of the parotid, possibly due to retrograde thrombosis along the facial vein; *b*, abscess of the joint of the left shoulder (case 13); *c*, abscess of the joint of the left hip, with a history of infection in the right hip; *d*, suspected abscesses in the right knee and ankle (not verified by incision) associated with obvious infection in the joint of the left elbow (case 12).

meningitis and cerebellar and cerebral abscesses, are of unusual interest. They will receive consideration under the section dealing with thrombosis of the cerebral veins. In 1 case a subaponeurotic abscess was found over the right temporo-occipital area in a 2 month old girl, who died of bilateral lateral sinus thrombosis and bronchopneumonia. The infection had extended in a retrograde fashion extracranially by way of the emissary vein of the right mastoid.

Thrombosis of the Cavernous Sinus.—Even in the presence of otitis media, the other possible causes of thrombosis of the cavernous sinus must be excluded before one can say that it is truly of otogenous origin. When the infection is secondary to disease of the middle ear it is, as a rule, also secondary to thrombosis of the lateral or petrosal sinuses. Of our 9 cases, the origin was open to question in 2, and the condition was very likely due to an accompanying infection of the sphenoid sinus or an orbital abscess. In the remaining 7 cases the condition was evidently secondary to thrombosis of the lateral sinus (via the superior petrosal sinus) in 3 and probably secondary to "primary" thrombosis of the superior petrosal sinus in 2.[37] In the other 2 cases thrombosis of the cavernous sinus was associated with meningitis and should probably be considered as more primary, in the sense that no other venous channel was found to be occluded. The thrombosis may have been secondary to the associated meningitis. In 1 of these cases there was associated labyrinthitis. While thrombosis of the cavernous sinus may be secondary to apicitis petrosa, such cases must be somewhat rare.

Thrombosis of the Petrosal Sinuses.—Occlusion of these smaller channels is usually secondary to thrombosis of the larger sinuses, particularly of the lateral sinuses. In the present series the superior sinus was involved 4 times (twice unilaterally and twice bilaterally), while the inferior sinus was involved 3 times (twice unilaterally and once bilaterally). In all these instances the thrombosis was associated with similar involvement in other channels and was probably secondary. Isolated thrombosis of these passages are rare, but a few cases of this nature have been reported.[38] Careful examination is necessary to discover these lesions.

Thrombosis of the Superior Longitudinal Sinus.—Otogenous thrombosis of this sinus is usually the result of retrograde extension of a thrombus in the lateral sinus, more frequently from the right side. In our series the superior longitudinal sinus was thrombosed in 7 patients with otitis media. In 2 cases it was consequent to thrombosis of the right and left lateral sinus and in 3, to one-sided thrombosis of the lateral sinus (the right in 2, the left in 1). In the other two cases

37. The reverse may, of course, be true, for thrombosis in the cavernous sinus may spread in the direction of the blood flow, i. e., into the superior and inferior petrosal sinuses. The rarity of primary thrombosis of the cavernous sinus makes this seem unlikely.

38. In a recent contribution, Brown and Bowman (Laryngoscope **43**:664 [Aug.] 1933) reported a case of thrombosis of the inferior petrosal sinus associated with thrombosis of the lateral sinus and the jugular bulb. In the opinion of these writers, thrombosis of the petrosal sinuses is secondary either to thrombosis in the lateral or cavernous sinuses or to infection in the petrous apex.

the process seemed to be primary, and, if this is true, the otitis was likely either merely a contributory factor or a coincidental condition.[39]

It is of importance to note that thrombosis of the superior longitudinal sinus following otitis media, whether primary or secondary, occurs almost invariably in infants suffering from dehydration and malnutrition. This will be considered in detail in a subsequent section.

Thrombosis of the Cerebral Veins.—Occlusion of the tributary veins is usually consequent to retrograde extension from an original thrombus in the larger channels. This appears to be a natural consequence, and one wonders why it does not occur more often. As the retrogressing thrombus formation reaches the mouth of the afferent vessels, occlusion of the venous flow results in stasis, which favors the formation of a thrombus which may be a direct extension from the original one or may be formed independently by a dissemination of infected particles along the static column of blood.

That local thromboses may occur in the cerebral veins without original thrombosis of the larger channels in the course of otitis media is a concept which seems to have been established by recent clinical observations. Such thromboses may be transient and followed by recovery, or they may develop into a more serious organic lesion. The earlier and intermediate stages are rarely observed at autopsy. The following case of primary thrombosis of a cerebral vein is of interest in this connection.

Case 15.—A Jewish man, aged 57, gave a history of lifelong otitis media, with a recurrence a few days before admission. Mastoidectomy was performed, and the dural plate was found to be extensively eroded and covered with granulation tissue which obscured the opening of a dural fistula. The patient died a few days later with symptoms of meningitis. Autopsy revealed generalized meningitis, an extensive subarachnoidal hemorrhage involving the sylvian cistern on the left and all the basilar cisterns and a localized subdural abscess, which resulted from a breaking down of protective adhesions about a local cortical ulceration. The extensive subarachnoidal and minor subdural hemorrhages were due to complete thrombosis of the inferior anastomotic vein on the left side. The lateral sinus was entirely free from any thrombus, although its walls were grossly inflamed. The infecting organism was Haemophilus influenzae.

These lesions are probably rare, although no data are at hand on which to base an accurate opinion. At any rate, we are concerned for the most part with secondary thrombosis of the cerebral and cerebellar

39. The possibility must be borne in mind that apparently unrelated and distant thrombosis may be a direct consequence of otitis. It is well known that multiple, apparently independent thrombi may exist in a lateral sinus. We believe that the same occurs in the cerebral veins, i. e., that isolated and distant thrombi may appear secondary to a thrombus in a lateral sinus. It is therefore possible that thrombosis may be initiated in the superior longitudinal sinus extending from the lateral sinus by way of the anastomotic veins.

veins due to retrograde extension from the larger channels. The groups of cerebral veins commonly affected are:

1. The inferior cerebral veins and the cerebellar veins—due to retrograde extension from lateral sinus thrombosis. Involvement of the inferior anastomotic vein may lead to widespread extension of the process to the dorsolateral surface of the cortex.

2. The superior cerebral veins—due to retrograde extension from the superior longitudinal sinus and influenced to a considerable extent by thrombosis of the lateral sinus on the involved side.[40]

3. The internal cerebral veins—usually due to retrograde extension from the lateral sinus (one or both) via the straight sinus.

Little attention need be given to venous thrombosis of the first two types, as the mechanism is obvious. A word might be added with profit concerning the third group, the internal cerebral veins. In addition to the distinctly primary group,[41] other instances occur in which the thrombosis is clearly secondary to retrogression of the thrombotic process from the lateral sinus into the veins of Galen via the straight sinus. One such case was observed in this series. In rare instances the condition appears to be primary, judging from the distribution of the thrombus, although it is associated with otitis media. Infection of the middle ear in these instances may be nothing more than a contributing factor and may be purely coincidental. The following case illustrates this possibility.

Case 16.—A well developed and fairly well nourished white boy, aged 1 year, was admitted to the hospital with a history of pertussis of three months' duration, which had terminated in bronchopneumonia. Four days before admission bilateral otitis media had developed. He died on the day of admission, and at autopsy primary thrombosis of the straight sinus, the great and small veins of Galen and the choroidal and terminal veins was found. The basal ganglions presented the characteristic hemorrhagic softening. The organisms found in the middle ears were pneumococci and Haemophilus influenzae.

The complications of thrombosis of the cerebral veins form an interesting group of lesions. It should be remembered that thrombosis of the lateral sinus per se is not necessarily a fatal or disabling lesion, even

40. This is emphasized by the fact that thrombosis of the superior cerebral veins may be unilateral and associated with extensive changes in the cortex of one side, the opposite hemisphere being free from any gross alteration. If complete thrombosis of the superior longitudinal sinus were alone responsible, the changes should be as marked in one hemisphere as in the other.

41. Thrombosis of the internal cerebral veins from whatever cause is a rare lesion, very few cases having been reported in the literature. They may be classified on the basis of etiologic factors into primary and secondary, as in the case of the other channels. The primary cases are usually found in women with chlorosis or in infants suffering from anemia and dehydration.

when the vessel is totally occluded; residual changes may be found years after an infection of the middle ear. Death is usually produced by one or more of a group of lesions, such as: a systemic infection due to the breaking down of an infected thrombus; consequent or associated intracranial lesions; commonly, meningitis; complicating myocarditis or nephritis, and, rarely, primary or secondary hemorrhage from the sinus itself. The intracranial lesions due to venous thrombosis are: subdural or subarachnoidal hemorrhage; subdural, cerebral or cerebellar abscess; hemorrhagic softening, and meningitis. Abscess of the brain secondary

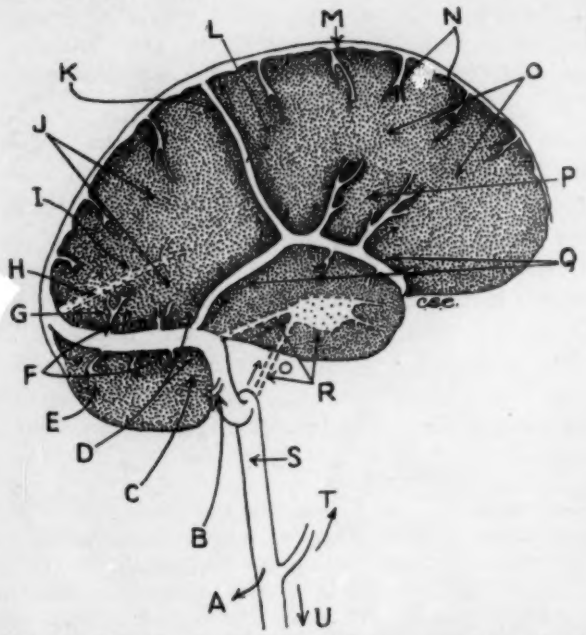

Fig. 6.—Diagram showing possible complications consequent to thrombosis of the lateral sinus. *A* indicates cervical abscess; *B*, thrombosis of the emissary vein, with an extracranial abscess; *C*, a cerebellar abscess; *D*, septic meningitis; *E*, thrombosis of the cerebellar veins; *F*, subdural hemorrhage and abscess; *G*, thrombosis of the inferior cerebral veins; *H*, abscess of the occipital lobe; *I*, thrombosis of the sinus rectus and the internal cerebral veins; *J*, hemorrhagic necrosis; *K*, thrombosis of the major anastomotic vein; *L*, abscess of the parietal lobe; *M*, thrombosis of the superior longitudinal sinus; *N*, thrombosis of the superior cerebral veins; *O*, subdural abscess and subdural and subarachnoidal hemorrhage; *P*, abscess of the frontal lobe; *Q*, thrombosis of the minor anastomotic vein and of the median cerebral vein; *R*, thrombosis of the superior and inferior petrosal sinuses and of the cavernous sinus; *S*, thrombosis of the jugular vein; *T*, abscess of the parotid gland; *U*, generalized infection and metastases.

to thrombosis of the lateral sinus, as the result of retrograde extension into the afferent veins, has received considerable attention in the litera-

ture and will be discussed later in another connection. Subdural abscess consequent to thrombosis of a venous channel has already been considered.

Subdural hemorrhage and hemorrhagic softening merit particular attention. It is not widely known that subdural hemorrhage may be the result of thrombosis of the cerebral veins. Such hemorrhages may occur in the same situations as subdural abscesses of similar etiology, and it is likely that the mechanisms do not differ greatly. With a virulent organism the toxins of which have a strong proteolytic action, the terminal portion of a tributary vein may be eroded to permit a local effusion of blood into the subdural space, and, at times, into the subarachnoid space as well. An abscess in the same location may result from a less virulent infection which permits a local exudative reaction to occur.

Five cases of subdural hemorrhage of otogenous origin have been recorded in the necropsies reported here, 4 of which were associated with and probably caused by thrombosis of the lateral sinus or of one of the other venous channels. The fifth was probably the result of meningitis; no thrombosis was found either in the sinus or in the afferent veins. In this case there was a diffuse hemorrhage over the surface and about the base of the left cerebral hemisphere. Both middle ears contained pus. In 1 of the 4 other cases, a small amount of subdural clot was found, apparently due to rupture of a thrombotic inferior anastomotic vein (case 15). The remaining 3 cases are briefly reviewed here.

CASE 17.—A white boy, aged 2 months, died with symptoms of meningitis after a short clinical course of otitis media. At autopsy the cavernous sinuses and the right superior petrosal sinus were found to be thrombosed. There was a diffuse subdural hemorrhage covering the cerebral hemispheres. There was also considerable hemorrhage mingled with exudate in the subarachnoid space. Smears from the middle ears and meninges revealed organisms resembling the pneumococcus.

CASE 18.—A white boy, aged 3 years, with a short history of otitis media, came to the hospital with obvious mastoiditis on the right side and thrombosis of the right lateral sinus, with metastatic foci in several of the larger joints. Mastoidectomy was performed, and the lateral sinus was explored after ligation of the jugular vein. The patient was doing well until the third day, when he pulled off the dressing; with it came the pack in the lateral sinus. He died of profuse hemorrhage. At autopsy a subdural hemorrhage was found beneath the basilar surface of the right occipital lobe. Its presence was evidently due to occlusion of the local inferior cerebral vein. The infecting organism was a streptococcus.

CASE 19.—A white youth, aged 19, died of slowly developing basilar meningitis consequent to chronic otitis media and mastoiditis. At autopsy a small thrombus was found in the sigmoid portion of the right lateral sinus, and in the adjacent subdural space there was a small blood clot covering an area about the size of a silver dollar (3.5 cm.).

These cases suggest that this type of subdural hemorrhage is usually a terminal condition and that it is not of vital interest from a therapeutic standpoint, as are recent or chronic subdural hemorrhages of traumatic origin.

When thrombosis of the small cerebral radicals led to the superficial cortical vessels, a different lesion was produced. Following widespread occlusion of these vessels, which usually occurred in children, the involved area assumed a dark red, "currant jelly" appearance (hemorrhagic encephalitis or necrosis). The involved part of the brain was swollen and softened. On cut section the cortex was found to be disintegrated and infiltrated with blood. Beneath and surrounding this

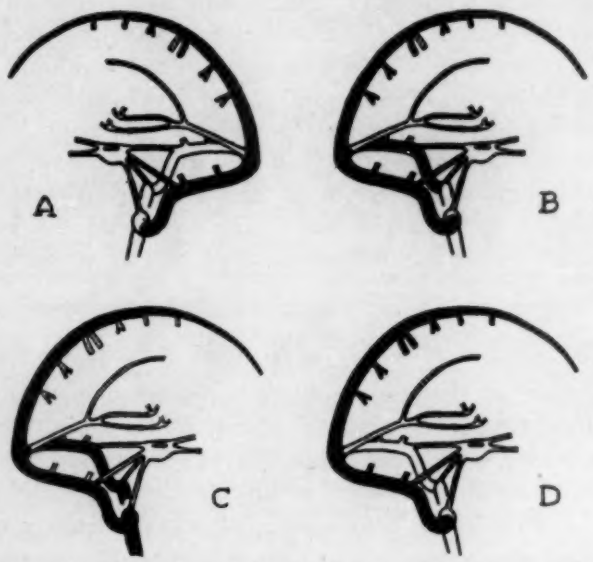

Fig. 7.—Drawing illustrating the extent of thrombus formation in 4 cases of widespread involvement of the venous channels: *A*, in a girl, aged 3 months, with associated septic meningitis; *B*, in a boy, aged 6 months, with hemorrhagic necrosis of the cortex of the right cerebral hemisphere and local bony necrosis and pachymeningitis about the genu of the right lateral sinus; *C*, in a girl, aged 4 years, with necrosis of bone to the right lateral sinus, bilateral otitis media, mastoiditis and multiple petechial hemorrhages of the cortex and white substance of the right cerebral hemisphere; *D*, in a boy, aged 1 year, with bilateral otitis media and mastoiditis, subarachnoid hemorrhage over the left frontoparietal region and the right frontal and temporal lobes and necrosis and hemorrhage of brain substance in the involved areas.

zone of necrosis a second zone marked with myriads of satellite petechial hemorrhages was found. In the cerebrum the involved area was extensive, but in the cerebellum it was limited and was often confined to a small spot at the outer portion of the anterior border of the lobe, where

it came in contact with the genu of the lateral sinus. Limited areas were also found in the cerebrum. In such cases these spotlike hemorrhages have been designated by some observers as areas of "hemorrhagic encephalitis," the forerunner of abscess of the brain. This lesion will be discussed under the section dealing with encephalic abscess.

Widespread Thrombosis of the Venous Channels in Infants.—The textbook classification of thrombosis of the venous channels usually includes two types: a primary, marantic or autochthonous type, which most commonly affects the superior longitudinal sinus, and a secondary or septic type, of which thrombophlebitis of the lateral sinus is a classic example. The group of cases considered here seem to constitute a combined marantic and septic type. Acute otitis media resulted in a local thrombus in the genu of the lateral sinus and changes in the quality of blood, and a slowed rate of flow consequent to malnutrition, anemia and dehydration favored an extensive retrograde development of the thrombus, with widespread involvement of the system of venous sinuses. The venous system which drains the vault was affected almost exclusively. The clinical aspects of this group of cases are of considerable interest, but they cannot be discussed here. The extent of the involvement of the venous system in the various cases in this series is indicated in figure 7.

INFLAMMATORY LESIONS OF THE LEPTOMENINGES

The reaction of the leptomeninges to infection in the middle ear or mastoid constitutes one of the most interesting subjects in the field of neuropathology. The term meningitis is at times so loosely employed from a clinical standpoint that it is difficult for a pathologist to know just what is meant by the designation. This is particularly true of the term localized meningitis. Numerous details of the pathogenesis of meningitis need to be elucidated, since, from a therapeutic standpoint, the lesion constitutes a major challenge to medical science. The study of its morbid anatomy has its limits of usefulness, although deductions may prove to be of value in understanding the course of the disease.

In considering the life history of lesions affecting the leptomeninges, two distinct groups of conditions are to be recognized: (1) reactive or toxic disorders, in which, for the most part, recovery is possible, provided that the original focus of suppuration is drained, and (2) a septic process due to invasion of the subarachnoid space by the infecting organism.

The Reactive Meningitides.—The pathologist has had little experience with this group of conditions, since their presence is usually detected by clinical examination aided by analysis of the cerebrospinal fluid. Whether one is dealing in this group of cases with varying degrees of

irritation, depending on the virulence of the organism concerned, or with peculiarities of the anatomic pathway of invasion is not yet known. Suffice it to say that in 1 case, in which there was clinical evidence of meningitis, a moderate increase of cerebrospinal fluid under elevated pressure but with little or no chemical or cellular change was found (meningismus); in another case there was an increase in the number of lymphocytes and in the content of globulin (serous meningitis), and in a third case there was such a predominant cellular response (sympathetic meningitis) that infectious meningitis was suggested, and differentiation could be made only on the basis of the absence of living organisms in the fluid.[42]

These acute reactions in the leptomeninges are probably produced by bacterial endotoxins which reach the membranes by way of the various avenues entering the intracranial space. Their mode of entrance is at present more or less speculative, and the alert clinician, in cooperation with the neuropathologist, should find here a field rich in possibilities. When a patient whose condition has been clinically diagnosed as one of this group succumbs to some other disorder, the brain should be critically examined for minor (meningeal) changes associated with the local alteration in the temporal bone. In this way alone will one come to an understanding of the changes in the soft meninges accompanying varying clinical evidences of reactions.

A rather interesting though rare lesion with a definite morphology has been attributed to the late effects of meningeal irritation—chronic adhesive arachnoiditis.[43] The influence of otitic infection in the pro-

42. It is obvious, from a review of the literature concerning septic leptomeningitis of otitic origin, that this diagnosis has often been made on insufficient grounds. When there has been a strong cellular reaction to adjacent inflammation, as is demonstrated by a high cell count in the spinal fluid, the condition has at times been considered meningitis in which recovery has taken place after drainage of the mastoid (and often perisinuous infection). This likely accounts for the high percentage of cures reported in some quarters. For example, Hinsberg (Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **23**:124, 1926) reported 32 cures in 137 cases of otogenous meningitis (23.3 per cent), and Schpajer (Zentralbl. f. Hals-, Nasen- u. Ohrenh. **16**:104, 1930) reported 17.1 per cent of cures in 111 cases. We agree that cures may result in rare instances, but during the last four years we failed to see a single patient recover after organisms were cultured from the spinal fluid. Within this interval 74 cases came to autopsy, and perhaps one-third as many more patients died on whom postmortem examination was not carried out.

43. Chronic thickening, with opacity of the arachnoid, has a variety of causes. The senile form is commonly observed at autopsy. Syphilis plays the etiologic rôle in certain cases which show varying degrees of change from a linear or patchy thickening along the larger cortical vessels to a complete clouding of the dorsolateral surface of the arachnoid. In another group, injury of the head seems to be the causative agent, the meningeal reaction perhaps being due to subarachnoid hemorrhage. In still another group no definite etiologic factor has been determined.

duction of arachnitis is difficult to decide. Horrax [44] reported a series of 33 cases in which thickening of the arachnoid was followed by cerebellar symptoms. Suboccipital exploration was undertaken in each case. In 5 of these patients the chronic course following otitis media was characterized by marked labyrinthine symptoms. In 1 case a cyst was found in the right lateral recess. In the verified cases of Horrax's series the condition was more or less generalized. There are other localized changes which frequently lead to the formation of cysts. These serous cysts in the cerebellopontile angle frequently give rise to the symptom complex described by Bárány.

In the series of 10,000 cases reviewed here, a single case of cyst of the cerebellopontile angle, apparently due to otitis media, was disclosed. It was mistaken clinically for a cerebellar abscess. The case was reported by Meyers [45] in connection with conjugate deviation of the head and eyes and was further mentioned by one of us as one of the many pathologic entities which may occur in the cerebellopontile angle.[46]

CASE 20.—A Mexican child, aged 1 year, died following mastoidectomy on the right side and suboccipital exploration for a presumed cerebellar abscess on this side. The symptoms were associated with acute bilateral otitis media. Suboccipital exploration revealed a large cyst in the right cerebellopontile angle. Autopsy showed that the locally thickened arachnoid had been walled off to form a large cystic cavity filling the cerebellopontile angle, resulting in characteristic distortion of the adjacent pons and cerebellum.

Viewed from a clinical standpoint, it seems strange that, with the frequency of meningeal reactions following otitis media, the formation of cysts is not encountered more often.

Septic Leptomeningitis.—In this series of 10,000 cases, 246 cases of septic meningitis were found. In 128 of these otitis media and other complications were present. In 7 of this group disease of the accessory nasal sinuses was also present, and the exact source of infection was therefore open to question. From these figures one might conclude that about half of all cases of septic meningitis which come to autopsy are due to otitic infections.

Of all the intracranial complications of otitis media and mastoiditis, septic meningitis is the most serious, for its mortality approximates 100

44. Horrax, Gilbert: Generalized Cisternal Arachnoiditis Simulating Cerebellar Tumor: Its Surgical Treatment and End-Results, Arch. Surg. **9**:110 (July) 1924.

45. Meyers, I. L.: Conjugate Deviation of the Head and Eyes: Its Value in the Diagnosis and Localization of Abscess of the Brain, Arch. Otolaryng. **13**:683 (May) 1931.

46. Evans, Newton, and Courville, Cyril B.: The Nervus Acusticus: II. Pathologic Conditions Involving the Eighth Nerve and the Cerebellopontile Angle, Laryngoscope **42**:432 (June) 1932.

per cent. In a review of autopsy records, particularly of cases in which a histologic study of the petrous bones was not made, one is more or less limited to a study of the morbid anatomy of the disease.

In attempting to classify meningitis from the standpoint of pathways of infection, Goerke [47] gave the following classification:

Classification of Otogenous Meningitis

A. Direct or Primary Meningitis (direct extension from the temporal bone)
  1. Hematogenous form, by way of the pial veins
  2. Tympanogenous form
     (*a*) By normal preformed paths
     (*b*) Through developmental preformed channels
     (*c*) Through pathologically developed paths
  3. Labyrinthinogenous form
     (*a*) By preformed paths
     (*b*) By pathologically developed paths

B. Indirect or Secondary Meningitis (by way of other intradural complications)
  1. Direct transmission from
     (*a*) Thrombosis of the lateral sinus
     (*b*) Subdural infection
     (*c*) Abscess of the brain
  2. Concomitant or symtomatic meningitis associated with thrombosis of the lateral sinus, subdural infection or abscess of the brain

This classification leaves little to be desired, if each case which came to autopsy could be properly pigeon-holed. However, when one is confronted with a number of lesions in a given case, one is somewhat at a loss to know which is responsible for the meningitis. We shall limit the discussion to the so-called primary and secondary types.

Direct or Primary Otogenous Leptomeningitis.—Of the 128 cases of unquestioned septic meningitis, 89 were of the primary type, according to Goerke's conception, since no other intracranial lesion was present. About half of these cases (48) occurred in the first decade of life (fig. 8). In 51 cases the meningitis was due to acute otitis and in 22 to chronic or recurrent otitis; in 16 the duration of disease of the ear was uncertain. In the acute cases, 35 patients were under, and 16 over,

47. Numerous classifications of otogenous meningitis have been proposed, each having its points of advantage and weakness, whether considered from an anatomic, pathogenic or bacteriologic standpoint. The absence of evidence indicating the route of infection in primary meningitis and the exact evaluation of the situation in secondary meningitis make any complex classification, such as that of Goerke, rather impracticable so far as observations at autopsy are concerned. The classification proposed by Hinsberg (Bericht über die von 1903 bis 1925 an der Breslauer Klinik beobachteten Fälle von otogener Meningitis, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **23**:124, 1926) presents the same difficulties, although his subdivisions, which are concerned particularly with postoperative meningitis, add to the clinical interest of the presentation.

15 years of age. In the chronic cases, 9 patients were under 15 years of age, and 13 were older. This shows the greater tendency for acute otitis media to result in septic meningitis in childhood. In the 51 cases occurring within the first fifteen years of life, the exudate was chiefly basilar in 25 cases, chiefly vertical in 10 and generalized in 15; in 1 it was localized to the dorsolateral surface of the left cerebral hemisphere. In the 37 cases in adults (aged from 15 to 70), the exudate was chiefly basilar in 17, chiefly vertical in 6, generalized in 13 and localized to the dorsolateral surface of the left cerebral hemisphere in 1. In 1 case the location of the exudate was not made clear.

In 6 of the primary cases there was associated purulent sphenoiditis or ethmoiditis, and again the question arises as to which infectious focus

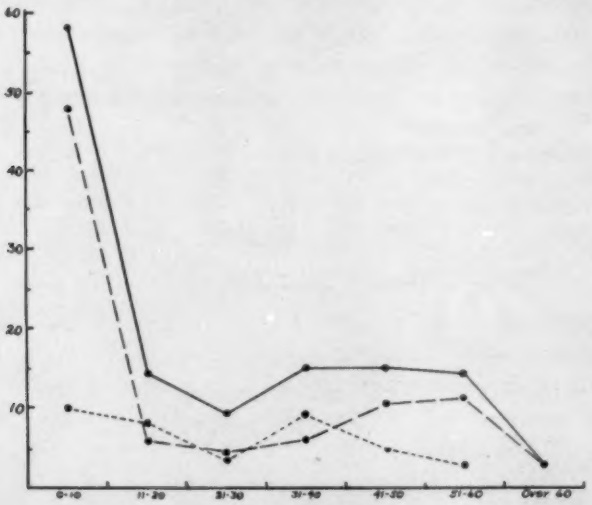

Fig. 8.—Incidence of otogenous septic meningitis in the various decades of life. The predominance of primary meningitis in the first decade is clearly indicated. The unbroken line indicates the total number of cases; the line with long dashes, the cases of primary meningitis, and the line with short dashes, the cases of secondary meningitis.

was responsible for the meningitis. This has already been referred to in an earlier section. In 13 of the primary cases there was erosion of the bone (in the tegmen tympani in 9, in 2 of which there were dural fistulas and in 1 pachymeningitis penetrans) and at the petrous apex in 4 (with extradural abscess in 2).

Indirect or Secondary Otogenous Leptomeningitis.—In the cases of indirect or secondary meningitis associated with other intracranial complications no attempt was made to trace the etiology in such detail as Goerke suggested in his classification. In many instances this would have been impossible, owing to the number of lesions. Listing the single

lesions aside from meningitis, there were 14 cases of abscess of the brain (in the frontal lobe in 3, in the parietal lobe in 1, in the temporal lobe in 6 and in the cerebellum in 4), 13 of thrombosis of the venous channels and 4 of subdural abscess. In 8 cases the lesions were multiple. There was a total of 39 cases.

In this group, 15 cases occurred during the first fifteen years of life and 22 during adult life (in 2 the age was not stated). The exudate in the first group was chiefly basilar in 10, chiefly vertical in 1 and generalized in 3; in 1 it was localized about the right temporal lobe secondary to an abscess in this lobe. In the second group the exudate was found to be chiefly basilar in 11 cases, chiefly vertical in 4, generalized in 3 and localized in 4. It occurred in the region of an abscess of the brain in the last group.

Of the entire group of 128 cases of meningitis, 89 were primary and 39 secondary. Of the total, 58 were found in the first decade of life. The distribution of this group according to age is shown in figure 8. In the entire group of cases, 65 of otitic infection were said to be acute, 41 chronic and 22 undetermined. The exudate was chiefly basilar in 63 cases, chiefly vertical in 20, generalized in 34, localized in 8 and not described in 3.

Concerning the bacteriology in this group of cases, there is little of value to add. Most of the bacteriologic diagnoses were made on the basis of a smear which is subject to various interpretations. On this basis, 74 cases showed pneumococci, 28 streptococci and 9 Haemophilus influenzae. Eight showed a mixed infection, and in 9 the type of organism was not stated.

OTITIC ENCEPHALITIS AND ABSCESS OF THE BRAIN

Abscess of the brain, particularly of otitic origin, has long continued to hold a prominent place in the field of intracranial suppurative lesions. This is due, in part at least, to the hope that it may prove to be curable—a hope not without possibilities, as the work of Sir William Macewen has so ably demonstrated. Few other lesions have received more attention in the literature; yet the pathologist still finds it easy to add to his store of knowledge from each specimen that comes to his hand. He should have as fellow students in the examination of a given lesion the attending otologist, the consultant neurologist and, perhaps, the neurosurgeon, if there is to be any widespread improvement in the treatment of this condition.

A consideration of inflammatory lesions of the brain consequent to otitis media should include more than the usual textbook description of a cerebral or a cerebellar abscess, although, to be sure, the morbid anatomy of abscess of the brain is concerned largely with its gross

description. As a prelude to the discussion of abscess of the brain, the problem of otitic encephalitis must have consideration.

Nonsuppurative Encephalitis.—The pathologic picture of otitic encephalitis is not well known, and there are gross defects in the conception of its pathogenesis. The term, used largely in a clinical sense, indicates a focal or widespread inflammation within the brain secondary to an infection in the middle ear or mastoid. It is not unlikely that some of the less marked neurologic manifestations are the result of a temporary alteration in the blood supply or a toxic irritation of the cerebral cortex and subcortex. The resultant lesion is evidently minor and transient, for complete recovery is the rule in these cases.

On the other hand, should there be an invasion of the brain by bacteria by way of either the vascular or the perivascular channels, an organic lesion is likely to result. It is possible for a hemorrhagic focus secondary to focal thrombosis to heal, with minimal damage, or even for a gross area of softening to heal without suppuration, with local shrinkage and residual gliosis. With a more virulent organism or a greater number of organisms, the primary softening or infarction breaks down, with the formation of pus. Limitation of this focus, with or without the formation of a capsule, depending on the size of the area, gives rise to a localized abscess. It is to be hoped that accidental discovery of the various intermediate stages of encephalitis will lead to a more complete knowledge of the pathogenesis of encephalic abscess. In this connection we shall concern ourselves primarily with the observations in the instances of otitic encephalitis which were encountered in this series of cases.

Three distinct lesions which might be included under the term encephalitis have come to our attention. One lesion occurred in the form of a large sharply circumscribed area in the right temporal lobe, which contained a granular blood clot; on microscopic examination this was found to be mixed with purulent exudate. This lesion seemed to answer the description of hemorrhagic encephalitis as described by some observers. It was obviously an early abscess in which gross suppuration had not taken place, and we therefore included it in the group of abscesses of the temporal lobe. The second group of lesions consisted of focal softening in which petechial or streaked hemorrhages were found. These areas were circumscribed and were found in the posterior part of the temporal region, along the lateral sinus in 1 case and in the lateral part of the anterior margin of the cerebellum, where this organ is in contact with the genu of the lateral sinus, in 3 cases. These lesions were evidently due to thrombosis of the local inferior cerebral or cerebellar veins. The third type of lesion consisted in an area of softening in the right parietal region, which on microscopic

examination proved to be an infarcted area with two small radiating streaks of cellular exudate. This was probably a more advanced stage of the second type of lesion, and, had time permitted, a gross local abscess would probably have formed.

Encephalic Abscess.—In the present series of 10,000 autopsies, 74 cases of abscess of the brain were found. Their source is indicated in table 2.

According to these figures, almost half (46 per cent) of the abscesses located within the substance of the brain follow otitic infections. This percentage is somewhat high according to the figures of Pitt,[48] who, in

TABLE 2.—*Source of Infection in Seventy-Six Cases of Abscess of the Brain*

Adjacent Infections:		
1. Otitic		35
Temporal lobe	17	
Parietal lobe	3	
Frontal lobe	3	
Occipital lobe	0	
Cerebellum	12	
2. Rhinogenous *		5
3. Infections of scalp, skull and spine †		4
Metastatic Infections		22
1. Lungs (abscess, 5; empyema, 5; carcinoma, 1; tuberculosis, 1)	12	
2. Heart (endocarditis)	5	
3. Liver (abscess)‡	3	
4. Bladder (cystitis)	1	
5. Teeth (apical abscess)	1	
Origin Undetermined		10
Total		76

* This includes 1 case each of carcinoma of the sphenoid, antrum and orbit.

† Abscesses following trauma to the skull are not included in this series. In such cases autopsy is performed by the coroner.

‡ This includes 1 case of pylephlebitis secondary to suppurative appendicitis, with multiple abscesses of the liver.

9,000 autopsies, found 56 encephalic abscesses, 18 of which were otogenous. In a series of 6,000 autopsies, Treitel[49] found 21 abscesses of the brain, of which 7 were otogenous. In 6,085 autopsies, Uchermann[50] found 35 cases of encephalic abscess, of which 14 were secondary to infection of the middle ear. No doubt, if the cases of traumatic abscess of the brain in which section is ordinarily done by the coroner were included in this series, our percentage of otogenous abscesses would be reduced to about that of the average.

48. Pitt, N.: Goulstonian Lectures on Some Cerebral Lesions, Brit. M. J. **1**:643 (March 22); 827 (April 5) 1890.

49. Treitel, L.: Ein Fall von multiplen otitischen Hirnabscessen, nebst einer Statistik, Ztschr. f. Ohrenh. **27**:26, 1895.

50. Uchermann, V.: Otitische Gehirnleiden, Ztschr. f. Ohrenh. **46**:303, 1904.

Of the 35 cases of otitic abscesses, 23 were cerebral and 12 cerebellar. This corresponds to the statistics in general. (Of 1,950 solitary otogenic abscesses, 1,281 were cerebral and 669 were cerebellar.[1])

Otogenous Cerebral Abscess.—Of the 23 cases of cerebral abscess, 17 were in the temporal lobe, 3 in the parietal and 3 in the frontal. Of the abscesses in the temporal lobe, 8 were found on the right side and 9 on the left side. This is not in accord with the conclusions of Lund,[51] who believed that abscess of the temporal lobe was the only otogenous intracranial lesion which tended to lateralize on the right. The average age of the patients with abscess of the temporal lobe was 34 years. Ten were males and 7 were females. Of the 3 abscesses of the parietal lobe, 2 were on the right side and 1 was on the left. The ages of the patients were 32, 37 and 45; all were males. Of the 3 abscesses of the frontal lobe, 2 were on the left and 1 was on the right. The ages of these patients were 9 months, 1½ years and 41 years, suggesting a tendency to occurrence in early life. Two of the patients were females and 1 was a male.

Cerebral abscesses consequent to otitis media and mastoiditis are the result of extension of infection into the intracranial space by one of two types of paths: (1) by way of contiguous structures, the infection extending through the tegmen tympani into the temporal lobe, and (2) by way of the venous system, through retrograde extension of the infection into the inferior cerebral or the inferior anastomotic veins to more distant points.[52] Unfortunately the cases of abscesses of the frontal lobe in this series did not come to our personal attention, and no mention was made in the brief description of the brain as to the state of the superficial cerebral veins; complicating meningitis presumably obscured any local

51. Lund, R.: Zur Klinik des otogenen Hirnabscesses, Ztschr. f. Hals-, Nasen- u. Ohrenh. **14**:341, 1926.

52. We place but little confidence in the theory of bacterial invasion of the brain by way of the larger arterial channels as a common cause of otogenous abscess. The possibility is granted, and in rare instances it seems to explain the mode of invasion. For the most part, however, distant abscesses are due to extension into the ramified system of external cerebral veins by way of the inferior anastomotic vein. The origin of abscesses of the frontal lobe secondary to thrombosis of this vein is clearly demonstrated in the cases of Bleyl (Ztschr. f. Hals-, Nasen- u. Ohrenh. **13**:16, 1925) and van Caneghem (Ann. d. mal. de l'oreille, du larynx **49**:340 [April] 1930). We further believe that this also holds true for abscesses of the frontal lobe which are on the side opposite to the ear from which there was a discharge. In these cases bilateral otitis media has existed, and the infection of the brain has been secondary to that of the ear and was minor and often undiscovered. One of the 3 cases of abscess of the frontal lobe in this series occurred on the side opposite to the ear from which there was an active discharge, as was the situation in the case described recently by Hill (Ann. Otol., Rhin. & Laryng. **39**:595 [June] 1930).

thrombophlebitis which may have been present. In this sense, 17 of our cases arose by contiguity [53] and 6 by possible retrograde extension into the vena anastomotica inferior. The location and relative size of these abscesses may be seen in figure 9.

It is well to emphasize the fact that there are wide variations in the appearance of cerebral abscesses. In computing the mortality in surgical cases it is of importance to note which type of abscess is considered. The classification of Piquet [11] seems to fill the requirements so far as the cases in this series are concerned. This classification merits a word of explanation. It is based essentially on the degree of limitation of the suppurative process. In the first group, that of limited localized abscesses, the cavity is sharply circumscribed from the surrounding brain, which may show a bordering zone of encephalitis of varying width. If the abscess is small and if a heavy wall of connective tissue has been elaborated, it is designated as encapsulated, whereas if it is larger and if the capsule is very limited in thickness or is wanting,

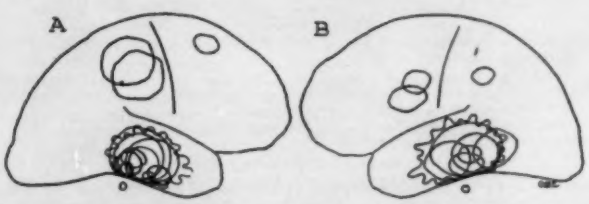

Fig. 9.—Drawings showing the location and relative size of the otogenous cerebral abscesses found in this series.

it is classified as nonencapsulated. In a second group of cases, classified as purulent encephalitis, a diffuse abscess of the brain is one in which an entire hemisphere or portion of it is involved, the affected area being transformed into a mixture of brain tissue, blood and pus. The second subtype in this group is classified as abscess of multiple foci, which is, perhaps, better designated as a diverticular abscess. This type of abscess has a cavity filled with pus and blood intermingled with shreds of brain tissue. Its walls are ragged and irregular, and there is a wide surrounding zone of encephalitis, in which other foci of suppuration are to be found. In the third group of cases, that of so-called acute nonsuppurative encephalitis, the condition is in reality not a postmortem lesion but rather one from which complete recovery may occur. If it progresses, however, it becomes the early stage of abscess of the

53. While in our series all the abscesses of the temporal lobe had apparently extended by way of the tegmen tympani or antri contiguous abscesses have been said to arise from extension through the bone over the posterior part of the semicircular canal or, rarely, through the squamous portion of the temporal lobe.

brain. The number of cases of each type, as found in this series, is indicated in table 3.

The following notes may be added in regard to the associated lesions in our cases. Of the 17 patients with abscess of the temporal lobe, 7 had meningitis alone (in 3 of which the abscess had ruptured into the ventricle), 1 had meningitis with thrombosis of the cavernous sinus and lateral sinus, 1 had an extradural abscess with thrombosis of the lateral sinus, 1 had meningitis with thrombosis of the lateral sinus, and 1 had septicemia. The remaining 6 had no other intracranial lesion, death ensuing from respiratory failure in the cases of the larger abscesses. Of the 3 abscesses of the parietal lobe, 1 was uncomplicated, 1 was associated with (or consequent to) thrombosis of the lateral sinus, and 1 patient died of septic meningitis following operation. All 3 patients with abscess of the frontal lobe had associated septic meningitis. These observations led us to agree with af Forselles [54] that otogenous encephalic abscesses

TABLE 3.—*Types of Cerebral Abscess*

1. Localized limited abscess		21
Nonencapsulated	10	
Encapsulated	11	
2. Purulent encephalitis		2
Diffuse abscess of the brain	0	
Diverticular abscess *	2	
3. Acute nonsuppurative encephalitis †		5

* Piquet used the term diverticular abscess or abscess of multiple foci to indicate a poorly outlined area of infection. It is not to be confused with abscesses with multiple foci, which may be well encapsulated.

† According to Piquet, this term includes a variety of lesions. The cases included here are described in the section on encephalitis, with the exception of one in which there was a large focus of hemorrhage and exudate, which we classified as an abscess.

are accompanied, for the most part, by other intracranial complications, although the figures which he referred to deal mainly with cerebellar rather than with cerebral lesions.

Multiple otogenous encephalic abscesses are infrequently encountered in reviewing the literature on intracranial complications. They may be multiple cerebral and cerebellar abscesses or simply multiple cerebral or multiple cerebellar abscesses. We found only an atypical example of the first group—a case of cerebellar abscess in which an attempt had been made to discover a cerebral abscess by probing. Along the needle track to the ventricle were found granulation tissue and pus formation, infection having been carried into the brain by the exploratory puncture. The 2 cases of multiple cerebellar abscess will be discussed in the subsequent section. Two cases of multilocular cerebral abscess were found: in the right temporal lobe in 1 and in the left frontal lobe in 1. In each case there was a central cavity with two smaller satellite abscesses.

54. af Forselles, Arthur: Der otitische Hirnabszess: Pathologie und Therapie, Acta oto-laryng. **10**:395, 1927.

Otogenous Cerebellar Abscess.—An interesting array of lesions may be included under the general term cerebellar abscess. The usual concept of this lesion is a more or less well defined cavity in the white substance of the cerebellum, lined with the usual pyogenic membrane. A study of a group of postmortem lesions gives one a very different picture of the disease and reveals some of the causes of the difficulties which are encountered in diagnosis and treatment. Many attempts have been made to classify the types of cerebellar abscesses. Müller [55] divided them into "interstitial" or encapsulated abscesses and "parenchymatous" abscesses, or those without sharp lines of demarcation. On the basis of location rather than of pathology, Friesner and Braun [56] divided them into superficial or cortical and deep or central abscesses,

TABLE 4.—*Classification of Otogenous Infectious Lesions of the Cerebellum* *

1. Localized subdural abscess		4
2. Focal hemorrhagic necrosis and superficial ulceration †		3
3. Intracortical abscess		7
(a) Miliary interfoliar abscesses	1	
(b) Small circumscribed intracortical abscesses	1	
(c) Large diverticular abscesses of the dorsal or ventral surface of the cortex	5	
4. Central abscess		5
(a) Small encapsulated abscess ("interstitial abscess" of Müller)	2	
Hemispheral abscess of primary type	1	
Peduncular abscess, secondary to otogenous basilar meningitis	1	
(b) Large limited but poorly encapsulated abscess	0	
(c) Diffuse nonencapsulated abscess ("parenchymatous abscess" of Müller)	1	

* In some cases more than a single type of abscess (as subdural and intracortical) existed, and therefore the total number of examples is greater than the number of cases.

† This is probably an inflammatory lesion, although true gross suppuration does not exist. Three such cases have come to our personal attention (discussed under the section on encephalitis). They were due to thrombosis of the local cerebellar veins.

mentioning in addition the occurrence of a local subdural collection of pus. On the other hand, Alexander,[57] was inclined to group the lesions according to their primary location and the direction of their spread, paying little attention to localization in the cortex or centrum. He classified them into (1) abscesses in the ventral portion of the hemisphere, tending to rupture on the ventral surface; (2) diverticular abscesses, working medialward to rupture on the dorsal surface; (3) abscesses in the region of the flocculus, migrating caudally; (4) abscesses of the vermis, and (5) multiple abscesses.

55. Müller, R., cited by Neumann, H.: Der otitische Kleinhirnabszess, Leipzig, Franz Deuticke, 1907, p. 10.

56. Friesner, Isidore, and Braun, Alfred: Cerebellar Abscess: Its Etiology, Pathology, Diagnosis and Treatment, New York, Paul B. Hoeber, Inc., 1916, p. 88.

57. Alexander, Gustav: Der otogene Kleinhirnabszess, in Alexander and Marburg: Handbuch der Neurologie des Ohres, Berlin, Urban & Schwarzenberg, 1929, vol. 2, pt. 2, p. 1435.

In an attempt to classify the types of lesions encountered in this group of cases, the standards of both localization and pathologic type have been utilized to characterize each group. For the sake of completeness and because clinically it cannot be distinguished from a true intracerebellar abscess, local subdural abscess has been included.

The various types of cerebellar abscesses are indicated diagrammatically in figure 10. Localized subdural abscess, while not a true cerebellar lesion in the strict sense, cannot be differentiated from one clinically; furthermore, it is commonly associated with one of the three types of intracortical abscess. Focal hemorrhagic necrosis, often resulting in a superficial ulceration, corresponds to localized hemorrhagic encephalitis secondary to thrombosis of the cerebral veins. It belongs

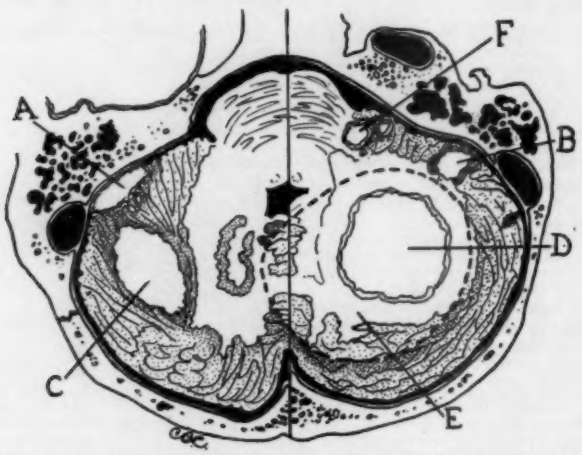

Fig. 10.—Drawing indicating the various types of otogenous abscesses found in the posterior fossa: *A*, circumscribed subdural abscess, with minute interfoliar abscesses; *B*, larger interfoliar abscess; *C*, intracortical abscess which may involve either the dorsal or the ventral cortex; *D*, central encapsulated abscess; *E*, large unencapsulated abscess involving one hemisphere and vermis; *F*, peduncular or floccular abscess secondary to chronic basilar meningitis.

in the group of encephalitic lesions and probably represents a transitional stage in the formation of abscess of the brain. Intracortical abscesses, from the miliary interfoliar variety to the large diverticular and dissecting lesions, are not encapsulated. The walls of the abscesses are formed by the pia mater compressed against the regional folia, a poor foundation for the proliferation of connective tissue. The smaller ones are in the strictest sense not surgical, and the larger ones are usually not successfully operated on. The central abscesses, located in the white matter of the cerebellum or the middle cerebellar peduncle, like those of the cerebral centrum, may be localized (limited or encapsulated) or diffuse, their size and the degree of encapsulation

depending on the virulence of the organism and the immunologic and tissue reaction of the host. The group of abscesses which follow protracted basilar meningitis is of interest. They are to be found in the middle cerebellar peduncle or adjacent flocculus and are secondary to the local accumulation of exudate in the cisterna pontis.

In the series under consideration 12 of the 35 otogenous abscesses of the brain were located in the cerebellum. Seven of the patients were males, and 5 were females. The infection in the middle ear was evidently chronic in 9 cases and acute in 3. Six abscesses were found on each side. The average age of the patients was 24½ years. These figures agree closely with the conclusions reached by Okada.[58] In regard to associated intradural lesions, 4 patients had septic meningitis as the single other lesion; 3 had thrombosis of the lateral sinus alone; 1 had meningitis and thrombosis of the lateral sinus, while 4 had no other intradural lesion.

For want of critical microscopic studies, one can only conjecture as to the pathways of infection in these cases. Judging from the findings at autopsy, 5 were due to direct extension through the bone and dura, usually associated with intradural adhesions and exudate; 2 were secondary to thrombosis of the lateral sinus; 1 was evidently due to chronic basilar meningitis; 1 was probably due to coincident labyrinthitis, while in 3 the pathway was uncertain or unknown.

Multiple cerebellar abscesses are not uncommon for three reasons: (1) owing to the formation of abscesses along the course of an infection which has spread between the folia, (2) owing to multiple thrombosis of the cerebellar veins and (3) owing to multiple points of invasion from a diffuse exudate about the peduncles. Furthermore, a local subdural abscess may be associated with any type of intracortical abscess. Multiple abscesses of the cerebellum were found in 2 cases—secondary to meningitis in 1 and to a local subdural abscess in 1. The abscesses were small in both instances and did not constitute surgical lesions.

A PROPOSED PLAN FOR THE STUDY OF POSTMORTEM MATERIAL IN CASES WITH INTRACRANIAL COMPLICATIONS

Since much of this material has passed through the Laboratory of Neuropathology in recent years a plan of study has been elaborated which has proved to be more and more successful in dealing with the pathologic material. The following outline for the study of the intracranial contents is suggested:

As these cases are of an infectious nature and cultural studies of the responsible organisms are important, embalming of the body is not desirable. The autopsy

58. Okada, W.: Diagnose und Chirurgie des otogenen Kleinhirnabscesses, Jena, Gustav Fischer, 1900.

should be performed as soon as possible after death before contamination with secondary invaders takes place.

The skull cap is removed in the usual way, care being used to avoid cutting the dura. The dura is then examined externally to determine its thickness and tension. The superior longitudinal sinus is opened and examined. The dura is next opened by incisions running parallel to the longitudinal sinus and extending 2 or 3 cm. on either side of it. This incision is carried from the occipital to the frontal region. A vertical incision is made, extending from the midpoint of the one just completed to a point somewhat behind the ear. This makes two triangular flaps on either side, which expose the dorsolateral surfaces of the cerebral hemispheres. The falx is severed anteriorly at the level of the sectioned frontal bone. The superior cerebral veins and pacchionian granulations are separated from the dura and sinus. This permits withdrawal of the falx, so that the cerebrum may be removed. This is accomplished in the usual manner, except that the cranial nerves other than the second are not cut but are left in place, and the midbrain is cut squarely across just above the pons. If this is carefully done, the two parts of the brain may be restored to their original relationships, should a photograph be desirable. The cerebrum is finally detached by severing the inferior anastomotic and the inferior cerebral veins on either side.

The structures in the posterior fossa are still in place and in their normal relationships for study of this region. The straight sinus, the lateral sinuses and the superior petrosal and cavernous sinuses are then examined by opening these channels with a sharp knife or with a pair of small scissors with the blades at an angle to the handle. When the sinuses and basilar cisterns have been examined, the structures in the posterior fossa may be removed by an incision of the tentorium close to its line of attachment along the petrous ridge and by section of the remaining nerves and the spinal cord.[59]

The internal structures of the cerebrum are studied, either before or after hardening, by a series of coronal sections. The cerebellum is sectioned by a series of cuts running vertically through each hemisphere at right angles to the anterior border. The remaining part of the brain stem is sectioned at right angles to its long axis to give the characteristic anatomic sections.

Dura Mater.—With this method of removing the brain, the dura can be studied in its entirety. Its internal surface should be examined for focal exudate or hemorrhage. The dura covering the anterosuperior

59. In the cases which came to autopsy in recent years we had the opportunity to examine the intracranial contents, while the temporal bones were removed and preserved for our colleagues in otology. Two methods of study of the temporal bones are possible, depending on the local circumstances. If means are available and interested persons are found, it is preferable to decalcify the bone and embed and section it histologically. If this is not the case, and if the most must be made of the morbid anatomy as seen at the time of necropsy, the following plan is advisable: The dura is carefully removed from the petrous portion of the bone, and the inner table of the anterosuperior surface is carefully chipped off, exposing the underlying groups of cells—those of the cochlea, the semicircular canal, the tympanum, the antrum and the superior part of the mastoid. By removing the petrous angle, the region surrounding the genu of the lateral sinus can be examined. This is followed by a study of the deeper mastoid cells. The crude method of driving a chisel through the tegmen tympani yields no information other than determination of the presence of pus in the middle ear and antrum.

surface of the petrous bone should be critically examined for extradural abscesses, discoloration or perforations, which may be very small. The dura should also be studied with the possibility of chronic lesions in mind, such as thickening and adhesions, both external and internal. In the posterior fossa the dura should be carefully inspected along the lateral sinus beneath the tentorium for local hemorrhage or exudate.

Venous Channels.—The venous sinuses should be studied while the dura is still in place, with the exception of the inferior petrosal sinuses and the genu and descending portion of the lateral sinus, which are examined after removal of the brain stem and cerebellum. It is highly important to search the genu of the sinus for small mural thrombi, which are easily overlooked. In a known case of thrombosis of the lateral sinus it is best not to open this channel but to remove it with the surrounding dura and embed it for microscopic section. In this way the relationship of the thrombus to the wall of the sinus is better preserved.

Leptomeninges.—The soft membranes of the brain should be carefully examined for exudate or hemorrhage, particularly when there has been a clinical history suggesting the reactive forms of meningeal irritation. Adhesions to the dura in the region of the tegmen tympani are important. When exudate is present, a smear and culture should be made, and its amount, color, texture and distribution should be described. In cases without meningitis the amount of free fluid in the leptomeninges should be noted.

Cerebral Veins.—Perhaps no other structure of the exterior of the brain has received so little attention in routine examination as the cerebral and cerebellar veins. Careful scrutiny is imperative in many cases of intracranial complications, in order to note any local or extensive occlusion. Tiny focal thrombi are indicated by grayish patches in the lumen of these vessels. The inferior cerebral, the inferior anastomotic and the cerebellar veins should be especially examined.

Cerebrum.—The surface of the cerebral hemispheres should be examined for any evidence of pressure, as flattening of the convolutions, dislocation or the effects of distortion. The presence and degree of hyperemia should be noted. The parts demanding particular scrutiny are the basilar surface of the temporal lobe lateral to the midbrain, the region along the lateral sinus where the inferior cerebral veins empty into this channel and, finally, the course of the inferior and superior anastomotic veins. A hand lens is of value in studying small lesions. Any focal lesion encountered in section of the brain should, of course, be described in detail, particular care being used to localize it accurately with regard to both the internal and the external structures. Photographs of gross lesions are highly desirable.

Cerebellum.—Gross lesions of the cerebellum are not likely to escape attention, with one possible exception—local hemorrhagic necrosis and superficial ulceration of the outer tip of the lobe. These lesions may be very small. Local discoloration (due to local hemorrhage) and disorganization should be given careful attention. In section of the cerebellum, special attention should be given to the brachium pontis, for small abscesses may easily elude observation in this structure.

It has been found highly desirable in our work to have mimeographed outline drawings made of the surface of the brain and of the vault and base of the skull to indicate the location, extent and, at times, the character of focal lesions of the brain or skull. This promotes more accurate work, and in reviewing autopsy records it has proved useful in rapid evaluation of a given case.

SUMMARY AND CONCLUSIONS

1. This study has for its basis a review of the protocols of 10,000 autopsies, special attention having been given to the intracranial complications of otitis media and mastoiditis. It is complementary to another study which deals with the same group of cases from a statistical standpoint.

2. Some of the difficulties in the evaluation of autopsy records are discussed. The matter of mistaking coincidence for consequence, the association of disease of the nasal sinuses with otitis media and the relationship of various intracranial lesions are emphasized.

3. Death in cases of otitis media is usually due to associated malnutrition or dehydration, consequential bronchopneumonia or diarrhea, septicemia or bacteremia or an intracranial complication. The highest death rate occurs in the first year of life, when intracranial complications are inclined to be less common. In some cases otitis media is an incidental finding and plays no part in the fatal issue of the case (terminal otitis).

4. Infection in the petrous pyramid has been studied, with particular attention to its intracranial complications. Of these, extradural abscess and meningitis are the most common. Thrombosis of the cavernous sinus and abscess of the temporal lobe are extremely rare, and unequivocal cases have not yet been described in the literature.

5. Among the dural infections, special attention is drawn to subdural abscess, and its classification is proposed on the basis of the cases found in this series. Dural fistula is also considered, with reference to the various intracranial complications with which it is associated.

6. The incidence and pathogenesis of thrombosis of the venous channels and consequent complications are discussed, with special consideration of extensive involvement of the entire venous system. This type

represents a combination of etiologic factors: infectious, chemical and mechanical. The distribution of metastatic foci secondary to thrombosis of the lateral sinus is also considered.

7. The chronic forms of arachnitis and their rôle in the formation of arachnoidal cysts is discussed among the various lesions of the leptomeninges. A consideration of the primary and secondary types of meningitis is also presented.

8. Otitis as a cause of encephalic abscess is illustrated by a table classifying the 76 cases found in this series of autopsies. Special emphasis is laid on retrograde extension of infection by way of the venous channels as a cause of abscesses of the parietal and frontal lobes. A classification of cerebral and cerebellar abscesses, based on their morbid anatomy, is suggested.

9. A method is suggested for removal of the brain and for a study of the intracranial contents of patients dying of complications of otitis media. The regions which should be carefully examined in order to avoid missing minor lesions are pointed out.

PERIARTERITIS NODOSA OF THE TEMPORAL BONE

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Although it has been stated that periarteritis nodosa has been seen to involve nearly every artery in the body (Longcope),[1] so far as we have been able to determine, no description exists of the pathologic changes produced by this condition in the temporal bone. In the case to be reported the vascular changes in the temporal bone have been carefully studied. Of further interest is the associated incidental finding of otosclerosis. Before presenting this case it may be advisable to give a brief description of the condition known as periarteritis nodosa.

While the term periarteritis nodosa (Kussmaul and Maier[2]) connotes an inflammatory lesion about the arteries with formation of nodules on the vessel wall, the condition is in reality an arteritis or a panarteritis involving some or all of the layers of the wall of the artery. While nodules on the vessel may be observed macroscopically, they are at times not visible to the naked eye. According to present authorities, the presence of macroscopic nodules is not a requisite for the establishment of the diagnosis.

Usually from the medium-sized to the smallest arteries are involved. In the early stages there takes place a marked infiltration of the vessel wall with leukocytes, mainly polymorphonuclears, but also lymphocytes, eosinophils and plasma cells. The additional changes consist of "fibrinoid" degeneration and necrosis of the media with deposits of fibrin, according to Fishberg.[3] In the reparative process a production of granulation tissue containing dilated capillaries and finally the formation of dense fibrous and scar tissue take place.

From the otologic service of Dr. Friesner and the laboratories of the Mount Sinai Hospital.

1. Longcope, W. T.: Periarteritis Nodosa with Report of a Case with Autopsy, Bull. Ayer Clin. Lab. of Pennsylvania Hosp., Philadelphia, 1908, no. 5, p. 1.

2. Kussmaul and Maier: Ueber eine bisher nicht beschriebene eigenthümliche Arterienerkrankung, Periarteritis nodosa, Deutsches Arch. f. klin. Med. **1**:484, 1866.

3. Fishberg, A.: Zur Kenntnis der Periarteritis nodosa insbesondere der Histiopathogenese, Virchows Arch. f. path. Anat. **240**:483, 1923.

The vessel is frequently occluded by a thrombus within the lumen. This may be attributed to the marked primary alteration of the intima or to the secondary extension of the lesion from the media. In addition, as a result of the destruction of the muscular layers of the media and of the elastica in the intima and adventitia, aneurysmal dilatations are produced. These aneurysmal dilatations at times rupture, and extravasations of blood take place.

These inflammatory changes are found in the vessels of the various organs of the body. According to Gruber [4] the frequency with which the organs are involved is in the following order: kidneys, heart, liver, intestine, mesentery, peripheral nerves, spleen, gallbladder, skin, brain and meninges and the sympathetic nervous system.

Schmincke [5] described the histologic changes in the nerves of a patient who clinically had had neuritis as a symptom of periarteritis nodosa. In addition, he reviewed the pathologic changes in the nerves described by previous authors. These consisted of degenerative phenomena manifested by spotty areas of pallor of the nerve fibers, clump formation of the myelin fibers and, at times, a disappearance of the nerve fibers. There was an increase in the nuclei of the cells of Schwann as well as formation of new axis-cylinders. It is interesting to note that these changes in the nerves occurred at some distance from the lesion in the blood vessels.

Goldstein and Wexler [6] recently reported the changes in the eye in a case of periarteritis nodosa.

Clinically the disease makes itself manifest as an acute, subacute or chronic sepsis with irregular temperature, marasmus and anemia. The blood cultures are negative. Severe gastro-intestinal and genito-urinary symptoms and polymyositis, polyneuritis or a combination of these are not infrequently present. However, nearly all of the organs may be involved, and the symptoms attributed to them may be thus provoked. Because of this multiplicity of symptoms the difficulty of correctly diagnosing the condition may be readily appreciated. Nevertheless, in recent years the physician is ever on the alert for this disease, and a correct diagnosis may be made at times by the process of elimination. In one of our two cases the diagnosis of periarteritis nodosa was seriously considered by one member of the attending staff, while in the other the diagnosis was established by the pathologic exami-

4. Gruber, quoted by Gohrbandt, Paul: Beiträge zur Pathologie des Periarteritis nodosa, Virchows Arch. f. path. Anat. **263**:246, 1927.

5. Schmincke: Ueber Neuritis bei Periarteritis nodosa, Verhandl. d. deutsch. path. Gesellsch. **18**:287, 1921.

6. Goldstein, Isadore, and Wexler, David: The Ocular Pathology of Periarteritis Nodosa, Arch. Ophth. **2**:288 (Sept.) 1929.

nation of the appendix removed at operation. It is interesting to note that Manges and Baehr [7] reported a case in which the diagnosis was correctly made from a mesenteric nodule removed at operation, while Gruber made the diagnosis from the examination of the gallbladder removed at operation.

The cause of periarteritis nodosa is not definitely known, but it is fairly well agreed that it is not a distinct etiologic entity but more likely a "characteristic symptomatic hyperergic reaction of the small and medium-sized vessel wall due to a nonspecific toxic infectious process" (Gruber [8]).

REPORT OF A CASE

History.—A. M., a man, aged 33, was admitted on March 11, 1931, to the medical service of Dr. B. S. Oppenheimer. He had had repeated sore throat since adolescence, but one month before admission he experienced an acute attack of tonsillitis with an associated cervical adenitis and a temperature of 103 F., soon followed in succession by pains in the various joints of the body. These symptoms continued intermittently up to the time of admission.

In the past year he had about ten attacks of distinct cramps in the epigastrium, nausea and vomiting, each attack lasting about one hour. There had been no symptoms referable to the ears.

Physical Examination.—The patient appeared acutely febrile, suffering from pains in the joints and in the epigastrium. A systolic murmur at the apex of the heart and a precordial friction rub were found. Tenderness but no rigidity was present over the lower part of the hypogastrium. A purpuric macular rash was noted over the flexor side of the thighs.

Both middle ears were essentially normal, except for a slight congestion of the vessels of the right drum. The hearing was normal to the spoken voice. Tuning fork and functional tests were not performed.

Examination of the Blood.—The white cell count was 17,000, of which 81 per cent were polymorphonuclear leukocytes. The hemoglobin was 86 per cent; the blood pressure, 140 systolic and 70 diastolic. The urine showed red blood cells and casts. Cultures of the blood taken on four different occasions were sterile.

Provisional Diagnosis.—The following were considered: (1) acute rheumatic fever with visceral manifestations; (2) intestinal vascular thrombosis; (3) erythema with visceral manifestations.

Course.—After admission to the hospital the patient complained of severe pain in the abdomen. The signs and symptoms were referable to the lower right quadrant. A diagnosis of hemorrhagic peritonitis secondary to an acute appendicitis was made. An abdominal puncture gave negative results. An appendectomy was then performed, and grossly the appendix was apparently normal. However, pathologic examination showed a necrotizing lesion in the media of the arteries, and the diagnosis of periarteritis nodosa was made. Signs of an acute nephritis with uremia, bronchopneumonia and myocardial insufficiency developed. Except for

7. Manges and Baehr: Periarteritis Nodosa, Am. J. M. Sc. **162**:162, 1921.

8. Gruber, B.: Zur Frage des Periarteritis nodosa mit besonderer Berücksichtigung der Gallen- u. Nieren-Beteiligungen, Virchows Arch. f. path. Anat. **258**: 441, 1925.

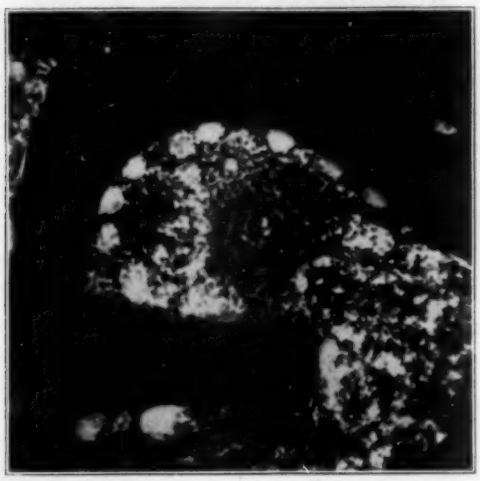

Fig.1.—Acute periarteritic changes in a small arteriole in the bone marrow of the petrous pyramid.

Fig. 2.—An artery in the region of the fossa subarcuata. The marked necrosis of part of the vessel wall (left) with resultant aneurysmal dilatation can be seen. A relatively normal artery is present to the right and above. *A* indicates adventitia; *M*, media, and *I*, intima.

a small hemorrhage in the right disk, the fundi were normal. The patient had a gradual downhill course and died about ten weeks after admission to the hospital.

Autopsy.—The following conditions were found: (1) generalized subacute periarteritis nodosa involving the heart, kidneys, gallbladder, pancreas, liver, lungs, diaphragm, stomach, intestines, bronchi, mesentery and spleen; (2) subacute fibrinous pericarditis.

Histologic Examination of the Temporal Bone: The smaller arterioles in the bone marrow of the petrous pyramid showed changes which were characteristic of periarteritis nodosa in the acute stages. Some showed subintimal proliferation with narrowing of the lumens, fibrinoid necrosis of the media and adventitia and perivascular cellular infiltration, mainly of the polymorphonuclear variety (fig. 1).

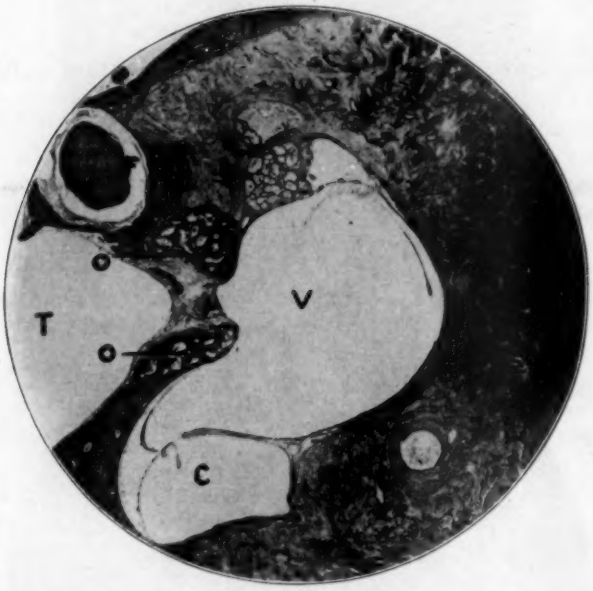

Fig. 3.—Vertical section through the vestibule, showing otosclerotic patches in the region of the oval window. *C* indicates cochlea; *F*, facial nerve; *O*, otosclerosis; *T*, tympanum, and *V*, vestibule.

In one of the vessels in the region of the subarcuate fossa there was a definite aneurysmal dilatation as a result of a hyaline degeneration and necrosis of the media; intimal proliferation and perivascular infiltration were also noted (fig. 2). The internal auditory artery showed no pathologic changes. In some of the vessels in the periosteum, particularly about the nerves and ganglion, the alterations usually found in arteriosclerosis, such as thickening of the intima and reduplication of the internal elastica, were noted. While the lesions in some of the vessels in this region were suggestive of periarteritis nodosa, the typical changes could not be demonstrated.

Three distinct patches of otosclerosis were noted in the labyrinthine capsule, one in the region of the oval window, the site of predilection (fig. 3), another in the modiolus and a third in the promontory at the outer insertion of the secondary

tympanic membrane. A careful study of the blood vessels in these otosclerotic areas was made, but the characteristic changes found in periarteritis nodosa were not seen. Except for the aforementioned findings, the middle ear and labyrinth were essentially normal.

COMMENT

As noted, otosclerosis was found histologically. It might be stated at this time that we have had one other case of periarteritis nodosa in which otosclerotic changes were found on histologic examination. This case was not included in this presentation because the periarteritic changes were not found in the temporal bone.

In order to determine any possible relationship between the two conditions a search through the histories of five other cases of periarteritis nodosa admitted to the Mount Sinai Hospital in the past four years was made. None of these presented any clinical evidence of otitic involvement. However, careful tests with tuning forks and histologic sections of the temporal bone were not performed in these cases so that the presence of otosclerosis could not be definitely ruled out. We also examined microscopically the temporal bones in five other proved cases of otosclerosis for the characteristic vascular changes of periarteritis nodosa, but none could be found. The material examined is insufficient to furnish evidence with regard to a possible relationship of these conditions.

SUMMARY

The clinical features and histologic alteration in the temporal bone in a case of periarteritis nodosa are described. Otosclerosis was an incidental finding.

Dr. Otani aided us during the course of the work, and Dr. B. S. Oppenheimer and Dr. I. Friesner gave permission to report the foregoing case.

DIVERTICULA OF THE THORACIC PORTION OF THE ESOPHAGUS

REPORT OF FORTY-TWO CASES

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Although the Zenker type of diverticulum is often considered to arise primarily in the esophagus, it is more correct to associate it with the lower portion of the pharynx. As a compromise it is sometimes described as a pharyngoesophageal diverticulum. Because of the fact that this type of diverticulum almost always produces difficulty in swallowing, it is recognized during life much more commonly than diverticula that are of true esophageal origin.

Esophageal diverticula are of much commoner occurrence than diverticula which arise from the pharynx, but because esophageal diverticula are usually of the so-called traction type, the apex of the sacculation points upward and rarely becomes of sufficient size to produce any symptoms referable to the esophagus. When these diverticula are found, it is almost always incidentally, at necropsy. Occasionally infection of the sac may occur, with perforation into a bronchus or into the mediastinum, resulting in the formation of an esophagobronchial fistula or mediastinal abscess.

A few diverticula of the thoracic portion of the esophagus have attained sufficient size to produce considerable distress. These have been recognized easily on roentgenoscopic examination. Also, smaller diverticula, which did not produce esophageal symptoms have been recognized when roentgenoscopic examination has been carried out as part of the routine examination.

In a review of diverticula of the thoracic portion of the esophagus, Heacock [1] found that 85 cases previously had been reported. To this series he added 2 cases that had come under his observation. In 1 of these there were multiple sacculations and the patient had experienced difficulty in swallowing. In the other case the sac was single and

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From the Division of Medicine, the Mayo Clinic.

1. Heacock, C. H.: Diverticula of the Thoracic Portion of the Esophagus, South. M. J. **23**:517 (June) 1930.

had not produced any symptoms. A third of the patients with deep-seated diverticula of the esophagus also had cardiospasm, but Heacock concluded that the majority of diverticula of this type are of little clinical value, their chief interest resting with the anatomist and roentgenologist.

In a recent report Barrett [2] found records of 115 cases, but he noted that large diverticula are extremely rare. He divided the diverticula into three groups: 1. In the first group he placed those that occur in the

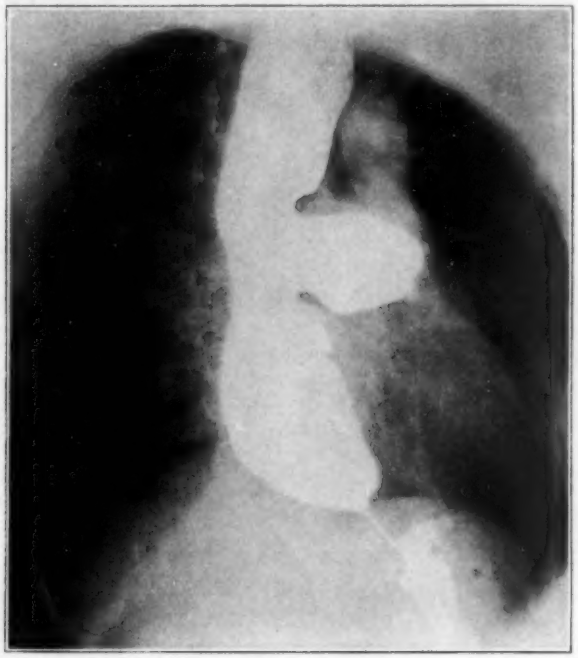

Fig. 1.—Cardiospasm associated with a diverticulum of the middle portion of the esophagus.

region of the left main bronchus, that are caused by inflammatory changes in proximity to the esophagus and that are described as of the traction type. 2. Diverticula of the pulsion type comprised his second group. They were formerly thought to be associated with spasm or stricture of the esophagus, but Barrett did not think that this association exists, because of the rarity of the occurrence of this type with cardiospasm.

2. Barrett, N. R.: Diverticula of the Thoracic Oesophagus: Report of a Case in Which the Diverticulum Was Successfully Resected, Lancet **1**:1009 (May 13) 1933.

3. The third group contains the pulsion-traction type of diverticula, in which the sacculation is originally produced by traction on a portion of the wall of the esophagus, and pulsion force gradually enlarges the pouch.

Raven [3] stated the belief that the majority of diverticula of the lower part of the esophagus that can be demonstrated during the patient's life are associated with cardiospasm.

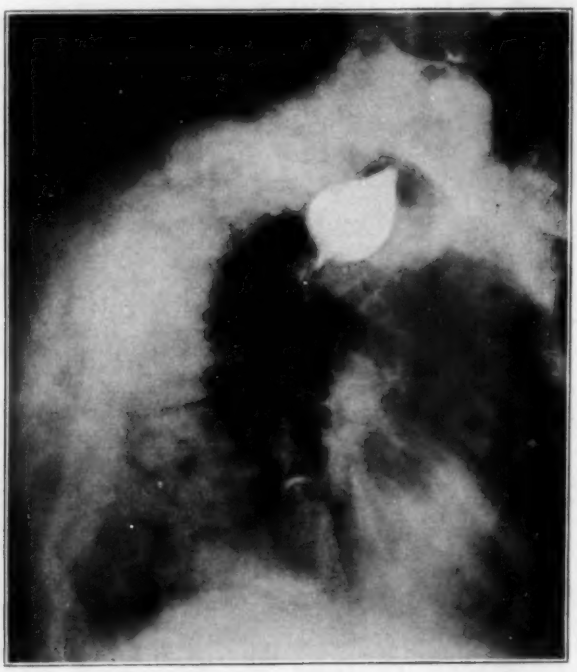

Fig. 2.—Carcinoma of the upper portion of the esophagus associated with a diverticulum above the cardia, as shown in figure 3.

Forty-two patients with diverticula of the thoracic portion of the esophagus have been observed during life at the Mayo Clinic, and afford the basis for this report. In reviewing these cases I have been impressed with the difficulty of evaluating symptoms and of associating them with the lesion in the esophagus.

Practically all of the patients had one or more symptoms referable to the alimentary tract, or they would not have been subjected to roent-

3. Raven, R. W.: Diverticula of the Pharynx and Oesophagus: Correlation of Pathological and Radiological Appearances, Lancet **1**:1011 (May 13) 1933.

genoscopic study. In 12 cases, it was decided that the symptoms experienced were not related in any way to the lesion found in the esophagus. In 12 other cases the symptoms were of such a type that it was questionable whether they could be attributed to the presence of the sacculation in the esophagus. The remaining 18 patients seemed to have symptoms that could definitely be associated with the abnormality in the esophagus. The symptoms in the entire group consisted of one or more of the following discomforts: dysphagia, epigastric pain or soreness, vomiting or

Fig. 3.—Diverticulum just above the cardia.

regurgitation, substernal distress, gas on the stomach, anorexia, belching, bloating, loss of weight, cough, chills and fever, heartburn, nausea, regurgitation of blood, and hiccup. The sex ratio was equal; 21 of the patients were men and 21 were women. The youngest patient was 27 years of age and the oldest 80 years. Fifteen of the patients were between the ages of 46 and 55. Thirteen of the patients had had previous respiratory infections; 3 of these had had definite tuberculosis. Eight had previously experienced injuries to the thorax. Eighteen of the diverticula were situated in the middle third of the esophagus, and the remainder

were in the lower third, usually just above the cardia. There were 2 diverticula in each of 2 cases.

The roentgenoscopic findings were of unusual interest; a summary of the reports follows: A diverticulum was present in 28 cases; cardiospasm associated with a diverticulum was present in 8 cases (fig. 1). One each of the following lesions was present: benign stricture of the esophagus associated with a diverticulum; carcinoma of the upper portion of the esophagus associated with a diverticulum at the cardia (figs. 2

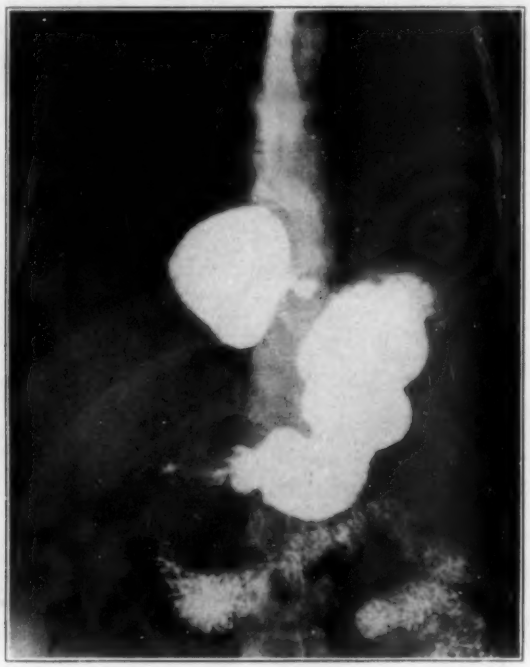

Fig. 4.—Two diverticula of the esophagus just above the cardia.

and 3); a diverticulum with a hernia of the cardiac end of the stomach through the esophageal hiatus in the diaphragm; carcinoma of the stomach with a diverticulum; obstruction at the pyloric end of the stomach with a diverticulum and duodenal ulcer with a diverticulum.

Esophagoscopic examinations were made in a number of the cases. In some the opening into the sac could be demonstrated; in others, the opening could not be identified, but secretion could be seen pouring from the sac into the esophagus, whereas in the majority of cases the esophagus appeared to be normal. In 1 case the sac had ruptured into

the left bronchus, forming an esophagobronchial fistula. Both of the openings of the sac could be identified on esophagoscopy and bronchoscopy.

In the majority of cases the symptoms were very mild, so that treatment was not instituted. Eight patients who had associated cardiospasm underwent dilatation with a hydrostatic dilator; 6 of them were completely relieved of symptoms. In 10 other cases large sounds were passed into the stomach; 4 of the patients were relieved of symptoms. The patient who had an esophagobronchial fistula was operated on, but the outcome was fatal. In 1 case the patient was instructed to wash out the sac twice each day, and this gave relief from symptoms (fig. 4).

Clinical Notes

TESTING THE HEARING OF THE PRESCHOOL CHILD

DOUGLAS MACFARLAN, M.D., PHILADELPHIA

The discovery of the handicap of deafness in school children led naturally to a desire to investigate the hearing of the preschool child. Up to this time, such efforts have been blocked by the difficulty of testing. No accurate tests have been invented heretofore.

The methods about to be described have been fully tried and offered to others in order to confirm their value.

TESTING THE HEARING OF CHILDREN TWO YEARS OF AGE

Until now, the hearing of these children has been determined by observing their response to clicking the fingers behind the ears and calling them by name and by noticing their attention to casual and familiar sounds. These observations are not, of course, quantitative tests.

The Test.—A buzzer audiometer is used. The sound is transmitted over a telephone circuit in which has been placed a decibel meter. There is a cut-off on the line. The child is trained, in the necessary number of sessions, to raise the hand when the sound is presented and to drop it when the sound is absent. The reaction is initiated by the examiner's raising the child's hand as the sound goes on and pulling the hand down when the sound is cut off. It is essential that the sound be loud enough to be heard.

When the child understands what is wanted and can make the response spontaneously, the decibel meter is put in use, and the loudness is gradually reduced until the responses are uncertain or inaccurate. The child must not see the cut-off being operated.

TESTING THE HEARING OF CHILDREN BETWEEN TWO AND SIX YEARS OF AGE

The present 4-*A* audiometer test is unsatisfactory for subjects of this age because the writing down of the numbers heard interrupts the attention to hearing. With some of the older children this may be obviated by having them recite the numbers. Group testing for children of from 2 to 6 years is impracticable. Individual testing only is satisfactory.

The Test.—A phonograph record using common monosyllables in the vocabulary of a young child is placed on a phonographic audiometer. A decibel meter is plugged into the audiometer on one side; the other side is led off to an ear phone. The speech comes through at a single level of intensity until the meter is turned down. The child recites what he hears. As the meter reduces the intensity, he begins to miss words and to misinterpret. The reading on the meter at this point is referred to the reading for normal hearing.

Dr. Crowden, of the University of London, has devised an ingenious scheme which further simplifies this test. He has made a phonograph record speaking about twelve commonly known names, such as dog, cat, door, house and ball. On a card placed in front of the child these names are illustrated. As he hears the

word he points to the proper picture on the card. The intensity of the sound is reduced as with the present 4-*A* audiometer record. The level of intensity at which words are missed is the index of the loss of hearing.

COMMENT

Feeling the need for concentration on this matter of testing the hearing of the very young child, I trust that these suggestions will be taken up by others who have been giving thought to the matter. I will be glad to cooperate with them in helping them obtain the records desired.

Certainly, since deafness has been found to be an important factor in the future of the school child, it is an obligation of the otolaryngologist to determine to what degree it exists in the preschool child.

1805 Chestnut Street.

Abstracts from Current Literature

Ear

BLOOD INFECTION FROM OTITIS MEDIA: ANALYSIS OF SIXTY-THREE CASES. E. J. G. GLASS, J. Laryng. & Otol. **48**:754 (Nov.) 1933.

An analysis of sixty-three cases of infection of the blood from otitis media is presented by Glass. The patients are divided into groups, and the comparative mortality rates are given. The following groups have a relatively low mortality rate: (1) children under 10 years of age, (2) persons with otitis media operated on during the first week, (3) persons in whom metastatic abscesses develop and (4) persons with hemolytic streptococcic infections treated with antiscarlet fever serum. The prognosis is bad: (1) in patients more than 40 years of age, (2) in patients with chronic suppurative otitis media with acute exacerbation, (3) if septicemia has developed before operation and (4) in patients showing subcutaneous hemorrhage or jaundice.

EDITOR'S ABSTRACT.

SEROUS MENINGITIS WITH CEREBELLAR SYNDROME DURING THE SUBSIDENCE OF CATARRHAL OTITIS. G. WORMS, Rev. d'oto-neuro-opht. **11**:418 (June) 1933.

In January, 1930, a man, aged 32, had an attack of catarrhal otitis media in the right ear, without eventual suppuration. Shortly afterward, crises, characterized by violent headache, vertigo, nausea and vomiting and loss of consciousness, appeared and had persisted for ten months. Between the crises the patient complained of headache, malaise, vertigo and obnubilation. Examination in January, 1931, revealed dulness, deficient memory, slow cerebration, lack of attention and rapid intellectual fatigue. Neurologic and all other examinations gave negative results except that the right tympanic membrane was slightly bulging in the posterior superior segment and was rosy, the hearing was diminished, and a roentgenogram showed a slight clouding of the right mastoid cells. On March 15, a slight papillary edema, more marked on the right side, was observed; neurologic examination revealed a left cerebellar hemisyndrome (hypermetria and dysmetria of the left leg, adiadokokinesis of the left arm and muscular hypotonia) and hyperexcitability of the left vestibular apparatus. Following two subtemporal decompressions, two weeks apart, and roentgenotherapy, recovery recurred. The interest of the case is in the serious intracranial complication induced by an apparently mild otitic infection. A focus of limited extent (abscess or tumor) in the posterior fossa is suggested, but the results of the decompressions eliminated this hypothesis and resolved the diagnosis into one of a slowly evolving meningeal dropsy.

DENNIS, Colorado Springs, Colo. [ARCH. NEUROL. & PSYCHIAT.]

THE MICROSCOPIC STRUCTURE OF THE AUDITORY OSSICLES AND ITS DEVELOPMENT. F. OESTERLE, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **135**:311 (July 22) 1933.

On the basis of a study of thirty temporal bones, Oesterle describes in detail the structure of each of the ossicles. He finds that the microscopic structure of the auditory ossicles exhibits considerable deviation from that of the other bones. Lamellar systems which form the building stones of the long bones have a subordinate rôle in the structure of the auditory ossicles. The nonlamellar, fine-fibered, strand-like medullary bone termed "strand-bone" (*Strähnenknochen*) by the author forms the main constituent of the ossicles. The fact that the strand-

bone is regularly found in all bones of the new-born suggests that it represents the ontogenetic (perhaps also phylogenetic) forerunner of the lamellar bone. The osteoblasts must apparently mature before forming the highly differentiated lamellar bone. Whereas in the other bones of the body the osseous tissue is constantly undergoing transformation in response to traction and pressure forces, in the auditory ossicles the embryonic type of bone persists. The auditory ossicles are in constant motion, reacting to sound waves even during sleep. Despite this continuous functional stimulus the ossicles exhibit slight transformation. In recent experimental analyses of the traction and pressure forces on the auditory ossicles, no correspondence between the lines of force and the structure of the ossicles could be found such as is the case in the substantia spongiosa of other skeletal bones. The reason for the lack of transformation must be sought in hereditary factors.

EDITOR'S ABSTRACT.

QUANTITATIVE DETERMINATION OF LABYRINTHINE FUNCTION WITH TOTAL POSTROTATORY NYSTAGMUS: SIGNIFICANCE IN OTITIS INTERNA. S. UNTERBERGER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **136**:248 (Sept. 21) 1933.

Unterberger states that the observation of a nystagmus and its strength and direction may indicate a decompensation but that it gives no information as to the actual excitability of the labyrinth in question. This is true of the postrotatory (as now evaluated) as well as the caloric nystagmus. While the actual excitability of the labyrinth cannot be judged by the postrotatory nystagmus obtained in the direction of the labyrinth in question or by the relation of the postrotatory nystagmic reactions to each other, it can be determined by the total postrotatory nystagmus value, which is obtained by the addition of the two postrotatory nystagmus values. This is possible because the total postrotatory nystagmus values do not depend, to the same degree as do the individual postrotatory nystagmus values, on the appearance of decompensation or compensation. The author determined the total postrotatory nystagmus on a large number of test subjects with normal and impaired labyrinths. Values under thirty seconds indicate a severe labyrinthine injury and values over sixty seconds a normal vestibular excitability. Values between thirty and forty seconds may be considered as slightly lowered, because they are practically never encountered in normal subjects, and values between forty and sixty seconds as normal or practically so, as they occur almost only in normal persons. On the basis of these determinations, important diagnostic conclusions can be drawn concerning the degree of vestibular excitability and the impairment of labyrinthine function. The results of rotatory tests were formerly often mistakenly interpreted; this can be avoided in future by also taking into consideration the total postrotatory nystagmus value which makes much more accurate diagnosis possible. The total postrotatory nystagmus is especially valuable for the differentiation of different forms of labyrinth inflammation with preservation of function, not so much as to their types as to the extent and the degree of the impairment of labyrinth functions. The author suggests the term "labyrinthism" for a group of cases characterized by the absence of a fistula, slight manifestations of decompensation and normal or practically normal total postrotatory nystagmus values.

EDITOR'S ABSTRACT.

ANATOMY AND GENESIS OF CONGENITAL ATRESIA OF THE EAR. F. ALTMANN, Monatschr. f. Ohrenh. **67**:765 (July); 917 (Aug.); 1042 (Sept.) 1933.

Altmann discusses congenital auditory atresia on the basis of a study of the serial microscopic sections and wax models of eight cases (thirteen temporal bones) and the valid cases found in medical literature. The structure often differs in individual cases. The term congenital atresia of the ear does not designate a single well characterized malformation but a whole complex of generally causally syngenetic malformations (owing their development to the same cause) associated in various combinations. They concern the posterior portions of

the two superior visceral arches and involve the structures of the middle ear in varying degrees. The cause of the development of this complex must be an injurious factor the influence of which has affected the whole anlage of the ear, particularly in the region of the external and middle ear. The author divides the malformation into three genetically similar groups according to their severity. In the first group a tympanic ring (even if poorly developed) and a rudimentary tympanic membrane exist. The tympanum exhibits only slight changes. The second group exhibits great polymorphism. The tympanic membrane is severely malformed or lacking, and the tympanum is usually diminished in size. There are two subdivisions, depending on the presence or absence of the tympanic membrane. Proliferation of parts of the second visceral arch (stylohyoid or tympanohyoid or a combination) can occur in both. In the absence of a tympanic membrane and with slight or no hyperplasia of the second visceral arch there is development of the squama temporalis and the floor of the tympanum, which reduces the defect. This development appears to be in inverse proportion to the proliferation of the second visceral arch. The third group comprises the most severe cases, in which a tympanum did not develop. This classification considers only the condition of the middle ear, but a certain parallelism between the degree of malformation of the latter and that of other parts of the ear exists. The termination of the teratogenic period is usually a stage of development in which the embryo is from 16 to 20 mm. in length. In the severe forms, however, it may often be that of an earlier period.

EDITOR'S ABSTRACT.

OTOGENOUS SUBDURAL ABSCESS IN A CASE OF APPARENT TRAUMATIC DEAFNESS: CONTRIBUTION TO THE THEORY OF CONCUSSION OF THE LABYRINTH. H. BRUNNER, Monatschr. f. Ohrenh. **67**:1021 (Sept.) 1933.

Brunner discusses the doctrine of concussion of the labyrinth in connection with the case reports of a woman and her husband deafened following an automobile accident. Ten years later the woman died of diffuse purulent meningitis resulting from a subdural abscess through acute exacerbation of chronic otitis. The development of the intracranial complication is discussed in the light of the necropsy. The author believes that the severe and lasting impairment of hearing which immediately followed the trauma is explained by the extensive bilateral otosclerosis which was disclosed at necropsy. This otosclerosis, which was not diagnosed during life, was probably the cause of a slight but insignificant baryecoia which had existed before the accident. Clinical experiences have shown the unfavorable influence of physical trauma and especially of the psychic trauma usually associated with it on the evolution of otosclerosis. While there is no reason for assuming the occurrence of concussion of the labyrinth, it cannot be ruled out, and the case cannot be considered as a proof that concussion of the labyrinth does not exist. This assertion (made by certain authors) is contradicted by the condition of the husband of this patient, who was in the same accident. In this instance deafness with caloric hypo-excitability developed on the right side after the accident. No indication of otosclerosis existed. What appeared to be a longitudinal fracture of the right temporal bone was seen in the roentgenogram, but it did not extend into the inner ear. Clinically, the unilateral deafness must be considered the result of concussion of the labyrinth. The author defends his theory that concussion of the labyrinth can occur without fracture of the bony labyrinth. It results from sudden compression of the endolymph and perilymph and the disturbed circulation in the labyrinth caused by a trauma. This is supported by Wittmaack's recent observation of a case of postraumatic deafness in which the atrophy of the organ of Corti and the macula sacculi, with their respective nerves, was the same as in the author's first case but in which no signs of otosclerosis existed. The following possibilities may be considered as anatomic bases for posttraumatic deafness in which the labyrinth is only temporarily or partially injured: (1) previously existing otosclerosis, (2) isolated fracture of the cochlea, (3) concussion of the labyrinth and (4) rupture of the cochlear nerve.

EDITOR'S ABSTRACT.

RADICAL OPERATION THROUGH THE EXTERNAL AUDITORY MEATUS. F. THIES, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:459 (Sept. 4) 1933.

After tracing the development of the radical operation on the ear through the external auditory meatus, Thies discusses his father's method and the results obtained with it in 1,500 cases. The first step in the operation is the excision of a triangular cutaneous flap from the membranous meatus. The base of the triangle lies in the tympanic ring, the apex in the region of the spina meatus. The anterior side of the triangle lies in the superior wall of the meatus and the posterior side in the posterior wall, near the inferior wall. The posterior wall is exposed as far as the individual case demands. If the tympanic membrane is to be preserved, the membrane is separated from the lateral wall of the tympanic recess before the wall is extirpated in its entirety with as much of the posterior wall of the meatus as is necessary. The operation is indicated if chronic otitis media has extended to the epitympanic recess, the antrum or the mastoid process and conservative therapy has failed, provided that an intracranial or sinus complication is not present and no abscess formation has occurred in the mastoid. The presence or absence of a cholesteatoma does not play a significant rôle. Of the 1,500 cases in which the operation was performed by the author's father, cholesteatomas were present in 11.5 per cent. Among the others perforation of the cranial fossa was seen in 8.9 per cent, disease of the semicircular canal in 1.9 per cent and involvement of the lower portions of the mastoid in 33.9 per cent. Usually all the spaces of the middle ear were involved. No fatality occurred. In 70 of 84 patients who returned for examination the ear was dry and the cavity left by the operation was well filled with epithelial tissue. In 12 cases there was a slight discharge, and in 2 cases there was an abundant discharge, which yielded quickly to treatment. In 41 cases hearing was improved, in 3 it was worse, and in the others it was unchanged. The author reports in addition 111 cases in which radical operations on the ear were performed by this method at the clinic of the University of Leipzig, with comparable results. These cases included 72 of cholesteatoma. This operation permits the surgeon to watch the important parts of the ear from the beginning and avoid injuring them. The operation is much less of a strain on the patient than the radical operation from the exterior and can be employed when a patient is too weak to undergo the latter.

EDITOR'S ABSTRACT.

Pharynx

SEROUS MENINGITIS OF ALLERGIC NATURE. J. H. SHELDON, Lancet **1**:798 (April 15) 1933.

A case is reported in which an attack of acute tonsillitis was followed after an interval of eight days by a generalized scarlatiniform rash, and by two groups of symptoms which developed concurrently: (1) a series of involvements of the larger joints, characterized by pain and effusion, the latter being especially prominent in the knees, and (2) serous meningitis, of sufficient severity to give rise to 4 diopters of papilledema. Both groups of symptoms subsided after the development of a peritoneal abscess caused by a hemolytic streptococcus; its drainage led to complete recovery. The diagnosis of hydrocephalus caused by serous meningitis was established by the normal character of the cerebrospinal fluid, its increased pressure and the subsidence of symptoms, including the papilledema, after two lumbar punctures. The nature of the effusion into the joints was not established by the puncture of a joint, but its complete subsidence in every case indicated that the fluid was certainly not purulent, and it was therefore assumed to have been serous.

BECK, Buffalo. [ARCH. NEUROL. & PSYCHIAT.]

EXTREME ADENOPATHY. L. DE MARVAL and R. G. PONS, Arch. med. d. hosp. Ramos Mejia **15**:87 (Sept.) 1932.

A patient with infectious adenopathy had an enlargement of all the upper lymph glands and a red swollen throat. The blood showed 14,000 white cells, with 78 per cent neutrophils. Glomerular nephritis eventually developed. In the differ-

ential diagnosis in such a picture lymphatic leukemia, agranulocytic angina, basilar adenitis, malignant lymphogranuloma and infectious adenitis must be considered.

SANFORD, Chicago. [AM. J. DIS. CHILD.]

RECOGNITION OF CHRONIC TONSILLITIS. W. KRAINZ and F. J. LANG, Monatschr. f. Ohrenh. **67**:1096 (Sept.) 1933.

In an extensive investigation for the purpose of finding an objective clinical sign of chronic tonsillitis which would be demonstrable in all cases of this disease, Krainz and Lang found that in all cases of chronic tonsillitis there are sensitivity to pressure in the tonsillar fossae and palpable hardening of the posterior side on palpation of the fossae from the exterior. Histologic examination of fifty tonsils in such cases disclosed, in addition to the well known picture of chronic inflammation of the lymphatic tissue and the crypts, thickening, cicatrization and induration of the capsule and trabeculae of the connective tissue, thickening of the vascular walls and the accumulation of plasma cells and other inflammatory cells between the fibers of the capsule and the trabeculae and around the blood vessels and nerves, as well as around bundles of muscles. Comparative examination of tonsils which did not exhibit sensitivity to pressure in the fossae showed less cellular infiltration and the absence of extensive cicatricial formations in the capsule and musculature. The objective clinical sign thus appears to be explained by the inflammation within the capsule and the muscle. These conditions are to be interpreted as the result of inflammation extending beyond the boundary of the tonsil. Despite the cicatricial contraction of the capsule, the muscular function is preserved. The objective sign here described is the major clinical sign in the recognition of chronic tonsillitis.

EDITOR'S ABSTRACT.

OZENA AND TONSIL. G. HALÁSZ, Monatschr. f. Ohrenh. **67**:1176 (Oct.) 1933.

Halász states that the internal secretion of the tonsil is the regional hormone of the mucosa of the upper respiratory tract. Genuine ozena is a disease of endocrine etiology which results from tonsillar hypofunction in persons with the organic predisposition which favors the development of ozena. Ozena may be cured with the subcutaneous injection of an organic preparation containing tonsillar extract and extracts from the thyroid and the thymus, organs in close correlation with the tonsil.

EDITOR'S ABSTRACT.

Larynx

PARTIAL RESECTION OF LARYNX IN CARCINOMA. M. J. CHARSCHAK, Monatschr. f. Ohrenh. **67**:1185 (Oct.) 1933.

In 240 cases of carcinoma of the larynx, Charschak performed only 9 partial resections of the larynx as opposed to 130 total resections of the larynx, but from these 9 cases he concludes that a partial resection of the larynx is possible and efficacious in certain cases. He thinks the restricted indications of most authorities may be extended and that partial resection may be employed in cases of carcinoma of the false or true vocal cords, even if they are immobile and if the tumor has spread to the interior portion of the other side.

AUTHOR'S ABSTRACT.

TRAUMATIC AMUSIA: REPORT OF A CASE. R. SCHILLING, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:568 (Oct. 14) 1933.

Schilling reports a case of amusia that occurred in a professional singer as a result of an automobile accident. The patient received a violent blow on the head, which caused profuse hemorrhages from the nose and the facial wounds, a severe bruise on the forehead, a hematoma of the larynx and severe headache. At first the peripheral disturbances—the hematoma of the larynx and paralysis of the crico-arytenoid muscles—dominated the clinical picture and sufficed to explain why the patient was able to sing only the lowest tones of the range formerly at

his command, and those only with great difficulty. Later, when laryngoscopic examination showed disappearance of the laryngeal symptoms and stroboscopic examination demonstrated normal vibration of the vocal cords, the patient's continued inability to reproduce correctly tones in the upper middle register or to repeat musical phrases sung to him and his lack of judgment concerning the quality of his vocal production indicated the existence of a disturbance of the central nervous system. The patient lacked acoustic control although hearing was normal. He was unable to think a high tone, and the effort to do this, as well as the actual production of tone, caused him headache. The diagnosis of a disturbance of the central nervous system was supported by the discovery, through interrogation, of early manifestations of aphasia and agrammatism. The author diagnosed the condition as mixed sensory and motor amusia of central origin. The unusual severity of the flow and the slow recovery (the patient had been treated for two years at the time of this report) suggest that a hemorrhage in the temporal lobe occurred in addition to a cerebral concussion. The therapy, based on the principle of sensory compensation, aimed at establishing new paths of innervation, especially for the sense of touch and of vibration, to replace the paths forgotten as a result of the injury to the central nervous system. This was accomplished primarily by isochronous "tone massage" with a special electric apparatus (*Tastenisochron*). A diaphragmatic support and other devices supplemented this therapy, and vocal exercises played an important rôle. This treatment has been employed for two years with gratifying results and gives promise of a complete cure in the near future.

EDITOR'S ABSTRACT.

SUCTION IN LARYNGEAL DIPHTHERIA. H. VON WILLEBRAND, Acta pædiat. **13**: 499 (June 6) 1932.

Following the example of Lynah, the author has applied suction in laryngeal diphtheria, using a curved metal cannula shaped something like an intubator and connected with a vacuum cleaner pump. His series of observations is small, comprising only ten cases, but it presents an encouraging decrease in mortality and in the number of cases of croup requiring intubation.

McINTOSH, New York. [AM. J. DIS. CHILD.]

Nose

RETROBULBAR NEURITIS AND DISEASE OF THE NASAL ACCESSORY SINUSES. WILLIAM L. BENEDICT, Arch. Ophth. **9**:893 (June) 1933.

In a consideration of retrobulbar neuritis and disease of the nasal accessory sinuses, knowledge of the position of various bundles of fibers in the nerve with reference to the retinal elements and their projection into the visual field is used as a basis for determining the situation of obstructions along the visual pathways. Homonymous anopsia and scotomas are of particular significance. In addition to defects of the field, the results of ophthalmoscopic examination of the fundus are important. Central visual acuity is an important item in the data for understanding the nature of lesions of the optic pathways.

Disturbance of vision constitutes the chief symptom of conditions which affect the optic nerves, tracts and radiations, and the cause of such disturbance may lie entirely outside the nerve. Anatomic anomalies, such as abnormally placed bony structures, abnormal blood vessels, cranial disturbances and deformities and tumors with direct or indirect pressure, may be the cause of a partial or complete atrophy.

Interruption of function of a portion of the optic nerve produces various changes. It is important to understand that the optic nerve will recover from the effects of injury sufficient to cause temporary total blindness. This phenomenon constitutes the basis for all forms of treatment for optic neuritis, and is of primary significance in a consideration of treatment for retrobulbar neuritis. The effect on vision by other circumstances, such as toxins, degenerative lesions and diseases

of the nerve itself, endogenous toxins associated with acute and chronic diseases and particularly diseases of the paranasal sinuses, is a second, but as important, etiologic factor.

In studying such a case, the author desires that special care be paid to obtaining a careful history of the onset and course of the visual disturbances. The loss of vision may extend slowly for several days. Simultaneous occurrence of symptoms in both eyes is rare, although one eye may be severely affected, whereas the visual acuity of the other may be only slightly diminished.

In considering the course of the visual disturbance, recovery in acute cases may be unusually rapid. In many cases central vision may remain stationary for days or even weeks. In others the loss of vision may be so gradual that when first noticed the defect, even in the peripheral field, may be well advanced. Permanent blindness may be reached only after weeks or months of slowly progressive expansion of a central scotoma. Recurrence may occur, of greater or lesser severity, at intervals of as long as ten years. Two cases were reported of recurrence after twenty-four years and after forty-seven years, respectively. Complete recovery may follow recurrence in such instances. Spontaneous recovery of vision has been seen in retrobulbar neuritis caused by multiple sclerosis and alcohol and nicotine poisoning, and in other cases of undetermined origin.

Much has been written on the anatomic position of the fibers of the optic nerve and their relation to the nasal accessory sinuses, from which one gains the impression that disease of the sinuses or changes in their structural development have a direct bearing on the function of the optic nerves through contiguity. The anatomic variations in the sinuses permit a variety of relationships between the sphenoid and ethmoid cells and the nerve, in its passage through the optic foramen and on to the chiasm. It has been intimated that disease of the mucosa of the accessory sinuses may be transmitted to the optic nerve through direct extension or by diffusion of toxic material along the blood vessels that traverse the region. The incidence of retrobulbar neuritis from a pathologic process in the nasal accessory sinuses is slightly higher in the United States than in Europe. On the other hand, it seems that multiple sclerosis is more frequent in Europe than in the United States. Another most important and relevant factor is the very high frequency of a pathologic process in the nasal accessory sinuses and the relative infrequency of retrobulbar neuritis. Most writers agree that multiple sclerosis accounts for the greatest number of cases.

De Grósz observes annually approximately 100 cases of retrobulbar neuritis, in 15 per cent of which the cause is nasal. Gifford found 3.5 per cent of cases with nasal etiologic findings in retrobulbar neuritis. Wiener, in 1931, stated: "I could report case after case where no suppuration was found in the nose, in which optic neuritis or retrobulbar neuritis subsided with shrinking of the mucous membrane of the nose. The visual acuity improves, the scotoma gets smaller or disappears, and the symptoms of the blur are dissipated." The author states: "When one considers the vast number of cases of severe suppurative sinus disease without visual symptoms, the connection between sinus disease and retrobulbar neuritis becomes much less credible. Even in the presence of dehiscences in the bony walls in either acute or chronic disease of the ethmoid and sphenoid cells lying near the optic nerve, the incidence of visual disturbances in patients seen in the Mayo Clinic is negligible. The transmission of inflammation from the sinuses to the optic nerve, either by direct extension, through the blood or lymph stream, or by toxins emanating from slightly thickened mucous membranes and diffused as noxious vapors, has received no convincing experimental corroboration."

He gives in support of his opinion an analysis of the etiology of 225 cases of retrobulbar neuritis at the Mayo Clinic in the following statistics: multiple sclerosis in 155 cases, pernicious anemia and nicotine in 14, diabetes in 14, alcohol and tobacco in 28, syphilis in 2, congenital amblyopia in 4, familial causes in 1, sinus disease in 1, postpartum hemorrhage in 1, plumbism in 2 and indeterminate causes in 3. In support of this he draws another conclusion relative to surgical intervention in the nasal accessory sinuses and the effect of this intervention. He

believes that the secondary traumatic results are of importance: "Operation on the nasal sinuses has two effects which have not been fully taken into account by those who advocate them for retrobulbar neuritis. Packing of the nose with cocaine and epinephrine for anesthesia produces, first, ischemia and then congestion of the membranes. Following the operation there is continued congestion of the mucosa of the sinuses and the adjacent tissues until healing is complete. If the operation has been sufficiently extensive, there is commonly a rise of temperature of 1 or 2 degrees F. from the absorption of blood, which, in effect, is autovaccination. These two factors are similar to the effects produced by injections of foreign proteins, and the results are parallel. It is my conviction that drainage of secretion from the paranasal sinuses is not the prime factor in the relief obtained by the operation, but that the hyperemia due to the packing and reaction of the operation and the inoculation by absorption of blood are the factors which are responsible for any improvement noted. This conviction is further supported by the course of many patients following operation. Operations on the sinuses are followed by quick improvement, but too often relapses occur shortly after operation because hyperemia has not continued long enough. . . . If a suppurative disease of the sinuses is obviously present, an operation should be performed for relief of the local condition, and additional measures employed to treat the retrobulbar neuritis, for even in the presence of infection of the sinuses, one cannot be sure that some other factor is not present. Operations on the sinuses, in most instances, probably do little harm. In many cases they do some good. The chief objection lies in the employment of an inadequate and unwarranted procedure, when better methods of treatment are available."

SPAETH, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

THE ACCESSORY NASAL SINUSES IN SCARLET FEVER. DONALD SMYTHE CHILDS, New York State J. Med. **33**:141 (Feb. 1) 1933.

It has not been generally taught that infection of the sinuses is a frequent accompaniment of scarlet fever, but in this study sinusitis was found in 91 per cent of 383 cases studied by roentgenograms. Seventy-nine per cent showed hazing, and 12 per cent were opaque. The most frequent lesion was bilateral hazing of the maxillary and ethmoid sinuses, which occurred in 30 per cent of the cases; the next most frequent was bilateral hazing of the maxillary sinuses (27 per cent). Pansinusitis was present in 9 per cent. Exploration of the sinuses was made in but 1 case, and no pus was obtained. Ninety cases were restudied, and in only 12 per cent was there evidence of improvement; in 50 cases there was definite evidence of progression or extension of the roentgenographic evidence. Mastoiditis developed in 33 cases, and in each instance infection of the sinuses was shown on the same side.

AIKMAN, Rochester, N. Y. [AM. J. DIS. CHILD.]

PROGRESSIVE LETHAL GRANULOMATOUS ULCERATION OF NOSE. J. P. STEWART, J. Laryng. & Otol. **48**:657 (Oct.) 1933.

Stewart describes a disease for which there is no definite name and the cause of which is obscure. In his opinion the most descriptive term is "progressive lethal granulomatous ulceration of the nose." Ten cases of this disease are reviewed. It is practically confined to the male sex, the ratio being 9 males to 1 female. Eight of the ten cases occurred in persons between the ages of 28 and 42 years. The pathologic basis is unknown. From the clinical and microscopic appearances, the author concludes that the condition is not a tumor but essentially a pyogenic disease, a chronic inflammatory process. Malignant granuloma is an unjustifiable designation. He finds no connection between this disease and Hodgkin's lymphogranuloma. It is not one of formation but of destruction, while the presence of granulation tissue may approve the term "progressive granulomatous ulceration." The clinical picture is one of progressive destruction of the nose, face and pharynx. It is characterized by a mild leukocytosis (14,800),

or a leukopenia (2,200), with the white blood cells in their normal proportions. There are prolonged and hectic fever and frequent and severe hemorrhages. The duration of the illness is from one to two years. The most marked feature of this disease is the complete absence of resistance of the patient to the infection. It must be differentiated from ulceration occurring in the nose due to the following diseases: syphilis, tuberculosis, malignancy, agranulocytosis, mycosis and myiasis group, yaws or frambesia, leprosy, rhinoscleroma, leishmaniosis, rhinopharyngitis mutilans (gangosa) and trophic postencephalitic ulceration. In six of seven cases, when bacteriologic findings were given, the presence of a streptococcus in combination with a staphylococcus was reported. As regards treatment, local applications proved unavailing. Treatment with radium was employed in two cases with indefinite results; but high voltage roentgen therapy promised more success and deserves further trial. Eight patients died of the direct effects of the disease, namely, sapremic cachexia and repeated hemorrhage. One survived for four months after local cure before succumbing to generalized sarcomatosis cutis, and one died from atypical "miners' phthisis" four years after recovery from the local condition. A review of ten cases collected from the literature is appended.

EDITOR'S ABSTRACT.

NOTES ON NASAL PLASTIC SURGERY. A. B. K. WATKINS, J. Laryng. & Otol. **48**: 809 (Dec.) 1933.

Watkins makes no pretense of covering the subject of nasal plastic surgery, but he discusses fractures of the nasal bones, the maintenance of internal nasal splints in position, external pressure splints, implants for saddle nose, septal causes of crooked noses, dislocation of the anterior portion of the septum, reproduction of symmetry after excision of portions of the nose and partial rhinoplasty. He presents his modification of the Carter splint. He uses a U-shaped splint instead of a straight one. One limb of the U is inserted into the nose, and traction is taken from the external limb. This produces the same result as if traction were taken from the inside limb. Owing to the difficulty of adjusting the pull so that it will be exactly at a right angle to the nasal bone, he has added a curved limb from the forehead to the splint, and from this limb traction from the U-shaped splint is taken by silk threads. The patient can breathe through the nose when the splint is in use, and if there is an external nasal wound the splint can be adjusted to prevent pressure on it.

EDITOR'S ABSTRACT.

CHRONIC DISEASES OF PARANASAL SINUSES AND THEIR RELATION TO CHRONIC LARYNGITIS WITH BRONCHIECTASIS AND ASTHMA. A. PERONI, Arch. ital. di otol. **44**:385 (July); 513 (Sept.) 1933.

Peroni states that the existence of a chronic catarrhal inflammation of a sinus is very frequent with hyperplasia of the mucous membrane. Often this disease is not recognized and is diagnosed as a chronic catarrh of the rhinopharynx. The author mentions the characteristics of drainage of normal and pathologic secretions in such cavities and their tendency to run into the larynx. He describes many cases of chronic inflammatory lesions of the larynx due to undiscovered inflammation of the sinus of the hyperplastic catarrhal type, the most frequent variety being posterior hypertrophic chronic laryngitis. Another type of laryngitis of sinusal origin observed by the author is the contact ulcer of the larynx. He reviews the anatomicopathologic changes in this disease, which was first described by C. Jackson and which is characterized by ulceration of the vocal process of the cords. Peroni also made a systematic study of sinusal conditions in 115 patients with bronchiectasis. Frequently these patients, especially children, were carriers of chronic sinusal disease, of both the purulent and the catarrhal type. The author admits that bronchiectasis and inflammation of the sinus may have their origin in the same cause, and also that bronchiectasis may take place on the basis of congenital bronchial malformation. In any case the author sustains the

importance of sinusal lesions in bronchial reinfection and in perpetuation of the bronchiectatic process, owing to the continual drainage of secretions into the bronchi. He points out the necessity of sinusal therapy as an aid to bronchial therapy. He discusses the various theories which attribute great importance to nasal lesions in regard to the genesis of asthma. He dwells on the idea that asthma is frequently caused by a bacterial allergy of sinusal origin. Peroni deals with the allergic origin of simple asthma and studies the different aspects of sinusal lesions in asthma. He refers to a number of patients carefully studied from both the allergic and the rhinologic point of view, primarily intending to estimate the importance of sinusal lesions in the general pathologic picture of asthma. According to the author, the lesions of the sinus found in 80 per cent of the patients, even in the absence of lesions in the nasal cavity, are nothing but a hyperplastic process arising from the mucosa itself. In view of the foregoing facts, the author maintains that neither hyperplasia of the sinusal mucosa nor nasal polypi are the cause of asthma, but an epiphenomenon of the allergic process. He states in conclusion that it is unwise to consider allergies of bacterial origin frequent, and, on the other hand, that it is wise to intervene only with the lesions that are an obstacle to breathing functions.

EDITOR'S ABSTRACT.

NEW NASAL SYNDROME AND ITS LATENT FORMS. CARLOS CHARLIN, Arch. de oftal. hispano-am. **32**:265 (May) 1932.

The author reports another typical case of nasal nerve syndrome dependent on inflammatory and other lesions of this nerve, first described by him, and in its complete form characterized by the symptomatic triad of inflammation of the anterior pole of the eye, rhinitis and orbital and periorbital neuralgia. He next calls attention to clinical variations; that is, more pronounced ocular lesions (iritis) with mild nasal symptoms (limited to hypersensibility, redness and increased humidity of the nasal mucous membrane); to other cases in which the nasal symptoms predominate, the ocular being easily overlooked and reduced to a slight corneal hypersensibility or a mild superficial keratitis or a cyclitis discoverable only by the slit lamp, and to still other cases in which the orbital neuralgic symptoms are the most marked, the ocular and the nasal symptoms being easily passed over.

A cocaine-epinephrine spray usually relieves the attack, but to obtain a permanent cure attention must be paid to the removal of the nasal lesions (ethmoiditis, deviation of the septum, hypertrophy of the turbinate bone) and to any general exciting cause (diabetes, syphilis) underlying the neuritis.

C. E. FINLAY. [ARCH. OPHTH.]

ETIOLOGY AND THERAPY OF VASOMOTOR RHINITIS. I. M. CHAJUTIN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **136**:3 (Aug. 24) 1933.

Chajutin states that study of the various theories of the pathogenesis of vasomotor rhinitis leads to the conclusion that it is an endocrinosympathetic syndrome based on a constitutional or an acquired inferiority of the endocrinosympathetic system. Many internal diseases, infectious and otherwise, may result in local organic changes in various parts of the sympathetic nervous system. The immediate cause of the rhinitis may be one of a number of factors. The author considers calcium iontophoresis a favorable attempt at causal therapy. During the last two years he has treated seventeen patients with vasomotor rhinitis with calcium iontophoresis. Thirteen of them were completely cured and have had no more attacks; the other four have occasional brief attacks but these are so slight that they consider themselves cured. The following method is used: Tinfoil electrodes (160 square inches) are placed one in front and one behind the shoulders, a flannel pad soaked in a 2 per cent solution of calcium chloride being placed under the anode, and flannel soaked in water or salt solutions being placed

under the cathode. A tourniquet is applied above the electrodes to produce hyperemia. A current of 40 ma. is applied for twenty minutes, with two interruptions to reduce the resistance of polarization. The patients receive this treatment for one month, every six months, as the effect lasts only that long.

EDITOR'S ABSTRACT.

GENESIS OF PERFORATING ULCER OF THE SEPTUM. G. KELEMEN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **136**:242 (Sept. 21) 1933.

Kelemen reports that during submucous septum resection performed on a woman aged 64 with severe purulent sinusitis a sharply circumscribed yellow spot the size of a lentil was discovered in the anterior portion of the cartilaginous septum. This was histologically diagnosed as beginning necrosis. The case shows that the necrotic process in a perforating ulcer of the septum can attack the cartilage alone, without at first causing any changes in the soft parts which cover it. The smooth healing after the operation further confirms this opinion.

EDITOR'S ABSTRACT.

CLINICAL OBSERVATIONS IN 150 CASES OF VASOMOTOR RHINITIS. L. HEDDERICH, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:429 (Sept. 4) 1933.

Hedderich states that a specific allergic cause is often not demonstrable in vasomotor rhinitis because of the vast number of possibilities, because a monovalent form of allergy may become polyvalent so that the patient also reacts to nonspecific stimuli and, finally, because in most or many cases allergic hypersensitivity is combined with sympathiconeurotic hypersensitivity. He discovered a specific allergen in only 30 of 150 cases of vasomotor rhinitis. Seven patients were infected with ascarides; the disappearance of the latter brought an improvement of the rhinitis in 5 cases, although the specificity of this disease could not be demonstrated. Among the nonspecific cases angioneurosis was seen in 18, neuralgia of the trigeminus in 2 and hyperthyroidism in 4; constipation was seen in 15 per cent and asthma in 6 per cent. Dysmenorrhea without a demonstrable organic cause was seen in one fifth of all the women. In the specific cases desensitization was employed. In the other cases treatment was aimed at correcting the internal disturbances, although their etiologic relation to the vasomotor rhinitis could not be demonstrated. In some of the cases of hyperthyroidism cure of this condition caused disappearance of the rhinitis. In the cases with constipation treatment of the latter alone did not influence the rhinitis. Constipation may be only a manifestation of the sympathetic neurosis which is at the base of the rhinitis. The sympathetic nervous system in its numerous functions is subject to many injuries; therefore, treatment of nonspecific vasomotor rhinitis must be individualized, and the hereditary constitution, the mode of life, previous diseases, the intestinal activity, the endocrine function and all the functions in the domain of the sympathetic nervous system must be taken into consideration. By reducing the irritability of the nasal mucosa it is sometimes possible to interrupt the vicious circle in which the patients with vasomotor rhinitis are caught. Local treatment of the nose is not advisable. Conservative internal therapy which is suited to the case, if applied consistently and long enough to bring about a change (*Umstimmung*) in the organism, affords the best prognosis.

EDITOR'S ABSTRACT.

Miscellaneous

USE OF HEADBAND IN TREATMENT OF SYMPTOMATIC NEURALGIC HEADACHES. SCHÖLLNER, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **136**:267 (Sept. 21) 1933.

Schöllner has obtained gratifying results in the treatment of neuralgic headache by the application of pressure to the areas of pain. For this purpose he devised a headband with two adjustable pads which can be centered directly over the painful

areas. The pressure is regulated by a fine screw adjustment, and its exact control is important. The minimum effective pressure should be used. The band is applied for from five to twenty minutes at a time. He used it in one hundred cases comprising twelve cases of suppuration of the nasal sinuses with supra-orbital neuralgia, forty cases of catarrh with neuralgia (including also cases in which there was only anamnesis of colds), thirty-eight cases of supra-orbital neuralgia without demonstrable sinusitis, nine cases of otitis with neuralgia and one case of dental disease with neuralgia. The headaches were cured in fifty-three of the cases, an improvement after each treatment was obtained in thirty-nine, in four the result was doubtful, and in four it was negative. The author discusses at length the theories of the nature of neuralgia in relation to the possible mode of action of his treatment. He thinks that symptomatic neuralgic headaches are produced chiefly by stasis through circulatory disturbances (Merings). The belt causes the stasis to disappear, thereby removing the pressure on the nerve endings and relieving the neuralgia. It is important not to apply too great a pressure, as this in itself will cause pain.

EDITOR'S ABSTRACT.

WITTMAACK'S OPERATION FOR BILATERAL PARALYSIS OF THE POSTERIOR CRICO-ARYTENOID MUSCLES. A. I. GESCHELIN, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **136**:348 (Oct. 14) 1933.

Geschelin reports a case of bilateral paralysis of the posterior crico-arytenoid muscles in which he employed Wittmaack's operation with satisfactory results, both as to speech and respiration. This operation differs from all others hitherto employed in that it does not aim at widening the rima glottidis at the level of the usual position of the vocal cords, but changes the position of the rima glottidis into an oblique one. This is accomplished by resecting all of the muscles attached to the muscular process of the arytenoid cartilage, thereby obtaining a lowering of the posterior end of one of the vocal cords. The author's case together with Wittmaack's two cases and two others makes five in which this operation was employed. All were successful. If the results are confirmed by further observations, this operation should become the one of choice in the treatment of bilateral paralysis of the posterior crico-arytenoid muscle.

EDITOR'S ABSTRACT.

FREQUENCY OF METASTASES OF PRIMARY MALIGNANT TUMORS IN THE FIELD OF OTORHINOLARYNGOLOGY. E. DUSSA, Ztschr. f. Hals-, Nasen- u. Ohrenh. **33**:405 (Sept. 4) 1933.

Dussa states that necropsy in 200 cases of malignant tumors seen at the otolaryngologic clinic disclosed metastases in 18 per cent of the cases. Carcinoma of the external ear shows a very slight, and sarcoma of the external ear, a considerable, tendency to metastasize. In the face, nose, nasal sinuses and maxilla, carcinomas as well as sarcomas rarely metastasize. This is especially true of carcinoma of the superior maxilla. Sarcoma of the oral cavity shows a tendency to metastasize, and this is also true of carcinoma of the tongue. Tumors of the epipharynx, mesopharynx and hypopharynx show an equal tendency to metastasize Metastases of tumors of the parotid gland occur frequently, with a predilection for the bones. Metastases of tumors of the exterior part of the neck do not seem to be rare, although it is impossible to determine what percentage is branchiogenous. Malignant strumas metastasize with great frequency, with a predilection for the lung. Metastases of esophageal tumors are relatively rare and usually occur in the liver or lung. Laryngeal carcinomas, with the exception of those of the vocal cords, metastasize more frequently than is generally conceded. Bronchial carcinomas have a strong tendency to metastasize, particularly in the kidneys. Cachexia appears to be rather rare in malignant tumors in the field of otorhinolaryngology.

EDITOR'S ABSTRACT.

Society Transactions

MASSACHUSETTS EYE AND EAR INFIRMARY, OTO-RHINO-LARYNGOLOGICAL SERVICES

EDITED BY PHILIP E. MELTZER, M.D.

Regular Clinical Meeting, Sept. 27 to Oct. 4, 1933

PRESENTATION OF SECTIONS OF THE ESOPHAGUS. DR. H. P. MOSHER.

My interest has been aroused by the findings of enlarged blood vessels beneath the mucosa of the esophagus. I have come to believe that these large blood vessels can come through the surface of the mucosa and start a superficial ulceration. The more I study the esophagus the more I believe that the glands are the means through which the superficial layers of the esophagus become infected and then the deep layers, and that this is one of the chief ways of causing superficial infection of this structure.

The slide which Dr. Pollock is to show now is of a section of the lower end of the esophagus, at the cardia, showing a myoma of the circular muscles. I have found five or six instances of this type of involvement. The myoma is always at the lower end of the esophagus. In this case the patient had arteriosclerosis with bronchopneumonia, and there was a certain amount of infection of the glands of the lower end of the esophagus. The frequency of such myomas has not been reported, but evidently they are fairly frequent, and they are the potential cause of obstruction in the esophagus.

Speaking of webs, first we found webs in the esophagus and then we found exostoses as a cause of webs. We had a series of such cases; then we did not find webs. The next slide concerns a patient from New Jersey, in whose esophagus there are striking spurs, with apparently a web opposite. Within a week or so she is to have an examination by the esophagoscope.

In practically all the cases of so-called cardiospasm which we have seen, or about 99 per cent, we found a narrowing of the esophagus which looked like a fibrous band at the opening of the crural canal. The next slide illustrates the condition of a patient who has the typical picture of cardiospasm, but when I passed the diagnostic bag, the esophagus dilated beyond normal, and there is no fibrous stricture.

ACUTE FRONTAL SINUSITIS WITH ABSCESS OF THE BRAIN AND MENINGITIS. DR. J. E. QUINCY and DR. G. B. FRED.

A man, 49 years of age, whom I saw on August 16, in the evening, said that he had been bathing in salt water eight days before, and that the following five or six days he was at work but suffered from a cold in the head. He had a slight nasal discharge and an indefinite headache. When he came to the hospital his temperature was 103 F. Local examination showed no tenderness or edema over either frontal sinus. In the right side of the nose there was thick creamy pus in the middle meatus. In the left side no pus was seen. The left middle turbinal was enlarged and showed some polypoid changes. There was no history of previous sinus infection. Pulse and respiration were normal. A roentgenogram was taken that night, which showed a definite cloudiness in the left frontal sinus and in both antrums. There was some difficulty in speech.

The following day the patient continued to have fever. There was no tenderness over either frontal sinus. Conservative irrigation of the nose with shrinking sprays brought down very little pus from the left side and more from the right.

I thought that he should be operated on, as aphasia was beginning to develop. He had increased difficulty in speech. He was right-handed. I saw him on Monday night and sent him here from the Baker Memorial Hospital on Wednesday. Dr. Fred operated Wednesday night. He opened the right frontal sinus first.

DISCUSSION

DR. G. B. FRED: This patient was very ill. He was admitted late in the afternoon and was operated on that night. A spinal puncture showed 3,500 cells, with 90 per cent polymorphonuclears. The neurologic examination gave negative results, except for the aphasia. The eyegrounds were normal, and the fields of vision, tested roughly, were normal. The neck was not stiff. Roentgenograms showed some chronic thickening in the left frontal sinus, but on account of the pus in the right nostril I decided to open the right frontal sinus first. There was no edema anywhere. The bone was thick and hard. On opening the sinus, there was a profuse amount of thick creamy pus. The lining of the sinus was thin. I removed the anterior and posterior walls and found no pathologic changes on the dura. Because the sinus showed pus under pressure, with no evidence roentgenologically, I extended the incision from the left eyebrow and opened the left frontal sinus. No pus was found. When the posterior wall was removed, a normal dura was seen. The ethmoid sinuses on the right were exenterated and were found to be filled with thick creamy pus. The patient's condition was not good, and he was taken off the operating table. The next day he became worse. The aphasia became more pronounced. The neurologic consultant advised exploration for abscess of the left frontal lobe of the brain. Accordingly, I incised the dura over the left frontal lobe and with the Cushing needle in about 3 inches (7.6 cm.) and directed in an upward and lateral direction, I found from 3 to 4 drachms (from 11 to 15 cc.) of thick creamy pus. A Mosher drain was put in. The patient died the next day.

At autopsy the brain showed a subdural abscess between the dura and the brain. There was also basal meningitis, with thick creamy pus. The sphenoid and ethmoid sinuses on both sides were filled with thick creamy pus. There was no abscess in the brain itself.

DR. H. P. MOSHER: What caused the aphasia?

DR. G. B. FRED: The subdural abscess. The condition of the patient was not good, and we believed that further surgical procedure was inadvisable. As may be noted, the brain is much indented over the left temporal region from the large amount of pus which was found there subdurally.

DR. H. P. MOSHER: The question comes up whether the patient could have been operated on earlier. He was very ill in the beginning. My question is whether more importance should have been placed on the aphasia and the patient operated on earlier. This is a puzzling case, and aphasia means trouble. The next point I wish to bring out is that a subdural abscess, by pressure on the brain, can cause all the symptoms of an abscess in the brain. Looking at the brain, one sees an indentation. In other words, the symptoms of pressure should not be attributed to an abscess in the brain; they can be attributed to an abscess outside the brain, pressing on it.

DR. G. B. FRED: The patient was Swedish and did not speak English well. We were not sure whether the difficulty in speech was true aphasia.

DR. D. C. SMYTH: The second day after the operation the patient's condition grew worse from minute to minute. Dr. Kubik saw him and stressed meningitis. He stated that the prognosis was hopeless, as the duration of the condition was so short. He thought of an abscess in the posterior part of the left frontal lobe. As it turned out, it would probably have been better to have gone in through a clean incision on that side, but as the man had basilar meningitis the case was hopeless anyway.

DR. H. P. CAHILL: The one thing that bothered us at the time was that the condition was of such short duration. We thought that we should find a markedly infiltrated brain with a true abscess of the brain. This case teaches a lesson in observation. In a case of this type, after the frontal sinus has been explored, a subtemporal decompression should be made, and the brain should be viewed from that area.

DR. C. T. PORTER: Dr. Cahill, I should like to ask whether, if the abscess outside did not produce the changes in the eye, there might have been papillitis if the abscess had been inside the brain.

DR. H. P. CAHILL: I think that, in a condition which developed so rapidly as this, one might expect changes in the eyeground as in a true abscess of the brain.

DR. H. P. MOSHER: May not changes in the eyeground be caused by a subdural abscess also?

DR. H. P. CAHILL: They may be. The changes which come on rapidly show more quickly, even from the day of infection, which in this case was from eight to ten days before examination. It was this that made us think of meningitis rather than intracerebral abscess.

PANSINUSITIS WITH ORBITAL ABSCESS AND MENINGITIS. DR. G. B. FRED.

A student, aged 21, went swimming on Saturday afternoon. On Monday an intense headache developed and the patient was admitted to the Peter Bent Brigham Hospital, where he was given nasal treatment. He did fairly well, but the next day definite exophthalmos of the right eye developed; there was little elevation of temperature. He complained of headache. A diagnosis of possible thrombosis of the cavernous sinus was made, and the patient was referred here. Examination showed him to be stuporous. Roentgenograms of the sinuses showed no abnormalities. There was no pus in the nose. A spinal puncture showed 900 cells, the majority of which were lymphocytes. The diagnosis was doubtful. We didn't know whether the condition was thrombosis of the cavernous sinus, abscess of the brain or extradural abscess, even at the time of operation. Even after consultation with physicians of the departments of neurology and the diseases of the eye the diagnosis was indefinite. The eyegrounds were normal. The patient had definite exophthalmos, and because his condition was becoming worse he was operated on.

An incision was made through the right eyebrow. On incision of the periosteum there was an escape of pus from between the periosteum and the bone, which accounted for the exophthalmos and the fixation of the eye. The bone was purplish. On entering the right frontal sinus, a large amount of pus and thickened mucous membrane were seen. The anterior and posterior walls of the sinus were then taken off. The bony walls were found to be firm, and a large area of the dura was seen to be thick and gray. There was no true pus on the dura. Because the right frontal sinus showed so many pathologic changes, with negative roentgenographic findings, I extended the incision and opened the left frontal sinus. There was no pus. The posterior wall was firm. When this was removed a normal dura was found on the left side. All this time the patient's condition was very poor. Artificial respiration was given, and the operation was stopped before we were completely satisfied. An incision had been made in the midline to the hairline, and the two flaps were sewed to the scalp. The patient was put to bed, and treatment was given for shock. Ten or twelve hours later his skin became cold and clammy, and his heart began to fail. At that time neurologic examination indicated an abscess in the posterior part of the brain on the right. But the patient's condition was too poor to permit another operation. He died the day after the operation.

Permission for autopsy was refused, but the dura was incised, and thick spinal fluid was obtained. We cannot say that it was pus, but it was not normal spinal fluid. After death roentgenograms of the lungs were taken.

DISCUSSION

DR. A. S. MACMILLAN: The roentgenograms of the lungs show what looks like a tuberculous infiltration of the periphery. This, of course, is a postmortem observation; a great deal more thickening may be expected in the hilar region, but not in the periphery of the lung, where there is a definite infiltration which looks tuberculous.

DR. G. B. FRED: A sister of the patient died of tuberculosis.

DR. H. P. MOSHER: Do you think that the tuberculosis had anything to do with the prognosis?

DR. A. S. MACMILLAN: There is a perfect history of complications following swimming.

DR. H. P. MOSHER: For the benefit of the third year students who are present, swimming can cause the worst cases of sinus disease and aural infection. I wish to congratulate Dr. Fred on his swiftness and sureness in searching for all the pathologic changes which were present.

DR. D. C. SMYTH: Dr. Fred did not speak of marked cyanosis along with the clamminess. After death not only was the dura tapped, but the cannula was put in; nothing was found in the brain.

OSTEOMYELITIS OF THE FRONTAL BONE. DR. F. L. WEILLE.

A man, aged 21, was admitted to the hospital ten days ago complaining of swelling of the forehead. He had been swimming about five or six weeks before. The day after, he had contracted an acute cold in the head. He had acute frontal sinusitis, which was treated in Rhode Island; a few days later the symptoms became severe.

The patient was operated on. An external operation was performed on the frontal sinus on the left side. A puncture wound was made into the frontal sinus on the other side. Apparently no provision was made for drainage into the nose.

The patient had a rather difficult time but gradually got better. Three days before admission here, the forehead began to swell; this was associated with vomiting and headache. A roentgenogram of the skull showed osteomyelitis extending 3 inches (7.6 cm.) upward on the frontal bone. The anterior wall of the left frontal sinus was shown to be a sequestrum by the roentgenogram, and the patient was operated on the same day. Under ether anesthesia, an excision of about the length of the anterior third of the cranium was made. It was made T-shaped in the usual way and extended backward for what was supposed to be 2 inches (5 cm.) above the area of infected bone. The work was done from the clean area forward. About nine or ten drill holes were made after the periosteum had been elevated and the flaps were turned outward. The frontal sinuses were cleaned out rapidly at the end of the operation, and the piece of bone was removed in a single section.

Before operation, a diagnosis of probable extradural abscess was made, on account of the vomiting and headache. The patient had only slight papilledema. Lumbar puncture showed a pressure of 350 mm. of water with 2 cells. An extradural abscess extended upward for about from 2 to 5 cm. from the superior border of the frontal sinus, and the posterior and anterior walls of the left frontal sinus were sequestrums. On the right side the posterior wall was intact.

The electric drill was used. By this means it is possible to drill a hole through the skull with rapidity and get to the dura with ease and without danger to the dura. There is a medium-sized drill with a cutting surface which is extremely effective. The drill is somewhat awkward to use, in that the axis about which the saw moves wavers slightly. The Gigli saw was also used. The bone was elevated with moderate difficulty after the dissection had been done, but there was no injury to the dura from either the sawing or the drilling of holes. In drilling, I observed, as Dr. H. Cushing says, that there is a different sound when the saw gets down to the dura. The frontal sinus bone was taken off rapidly, and on the

right side the dura was found to be very adherent laterally. A small nick was made in the dura with rongeurs; the other instruments were not used. The wound was iodized and packed. Following the operation the patient had an uneventful convalescence.

For forty-eight hours after the operation the temperature was normal. At this point the dressing was changed, and the temperature rose, then gradually fell. A roentgenogram taken a few days ago revealed no extension of the osteomyelitis.

DISCUSSION

DR. L. G. RICHARDS: Did the longitudinal sinus stick to the bone?

DR. F. L. WEILLE: Yes. Dr. Fred separated the bone from the longitudinal sinus. The bone was very adherent over the entire area of underlying dura.

FURTHER REPORT: Since the operation the wound has slowly cleaned up, except on the right bone edge. A roentgenogram showed no progression. Within the last two or three days the right bone edge began to granulate properly. The granulations, which were red and flabby, are now beginning to shrink down. We are keeping the dressing soaked with hexylresorcinol, S. T. 37.

DR. F. L. WEILLE: The temperature has been normal for about two weeks. The patient is eating and sleeping well and is feeling excellent.

DR. G. B. FRED: Would it be advisable to clean out the ethmoid sinuses and, if so, when?

DR. F. L. WEILLE: The patient had no disease in the ethmoid sinuses. The disease was always in the frontal sinuses. The question has been raised whether the ethmoid sinuses should be exenterated now. The purpose is for providing drainage into the nose.

DR. H. P. MOSHER: That is not the intention. The wound is supposed to granulate.

DR. F. L. WEILLE: Dr. Garland believes that the ethmoid sinuses should be exenterated.

DR. H. P. MOSHER: If the wound granulates, that is the end of it. I should delay operating on the ethmoid sinuses.

DR. P. E. MELTZER: In a patient who is doing so well, I should close the wound over on one side for the cosmetic effect.

DR. H. P. MOSHER: The difficulty is that he is going to bleed, and he is rather pale. If this could be done without much bleeding, there would be no objection, but I should leave him alone for a while. This case was done "according to Hoyle." It was fortunate that the whole of the forehead came off in one piece; that, I believe, is the method to be used in these cases. As for the drill, Dr. Garland and I worked that out together, starting with Dr. Cushing's drill. This is the third case reported today in which swimming was responsible for the condition.

DR. D. C. SMYTH: The use of the drill shortened the operation. I think that both physicians worked less than two hours. While the dura was torn there was no escape of cerebrospinal fluid, so I don't think that the dura was torn through.

EPISTAXIS. DR. P. J. ROSS.

A 14 year old girl was admitted to the emergency ward from a hospital in Northampton. Twelve weeks before admission she had severe epistaxis following swimming. The bleeding area in the nose was cauterized, but she had repeated hemorrhages, and one month later was admitted to the hospital because of anemia. While she was there, very extensive studies of the blood were made. The bleeding and clotting time were normal. The red blood cell count varied between 2,000,000 and 3,000,000. The platelet count rose from 500,000 to 750,000. Since coming here she has had no serious hemorrhage. The nose has not been packed. We were not able to find a bleeding point, but occasionally there is a little streaking of the

pharyngeal wall in the morning. Roentgenograms taken two days ago showed pus in the right antrum. The patient said that while she was in the hospital in Northampton something was left in the nose for twenty-four hours and then removed. I don't know whether the bleeding is caused by the infection in the antrum or whether it is secondary to the treatment. The red blood cell count has been from 2,221,000 to 2,700,000, with hemoglobin 45 per cent. There is no history of bleeding in the family. There is no bleeding point. The membrane is very pale.

DISCUSSION

DR. H. P. MOSHER: What are you going to do for her?

DR. P. J. ROSS: I want to know what to do about the infection in the antrum.

DR. G. B. FRED: Has any definite condition of the blood been diagnosed?

DR. P. J. ROSS: No.

DR. L. G. RICHARDS: Following severe bleeding, we always suspect that the shadow in the antrum may be blood and not pus.

DR. P. J. ROSS: While in Northampton, she was given a diet of very high caloric value, with iron and ammonium citrate, but the blood picture showed no improvement.

DR. H. P. MOSHER: I should like Dr. Smyth to commit himself.

DR. D. C. SMYTH: I saw the patient yesterday. She has been bleeding intermittently for twelve weeks. I should like to have Dr. MacMillan tell us whether the condition is active purulent sinusitis or a new growth or what it is. If we are dealing with an acute suppurative condition of the antrum, we should open it.

DR. A. S. MACMILLAN: There is too much reaction in the bone for mere hemorrhage. The malar bone on that side is more dense than on the other.

DR. D. C. SMYTH: If the nose has been packed and repacked, sinusitis could develop.

DR. H. P. CAHILL: I wonder if, while the patient was in Northampton, any cause for the anemia was found. She has bled little while here.

DR. P. J. ROSS: The history was that she had had several severe hemorrhages one month before entering the hospital in Northampton. Examination of the blood showed anemia; the gastric analysis gave normal results. The physicians believed that she had a slight kyphosis of the lumbar spine, also that there was no dyscrasia.

DR. H. P. CAHILL: Would a new growth give a shadow like that?

DR. A. S. MACMILLAN: The bony wall has not been destroyed.

FURTHER REPORT: A radical operation on the right antrum was performed four days later. A blood clot and thickened polypoid membrane were found in the antrum, as well as a cherry red polyp in the middle meatus. The patient had a blood transfusion two days before the operation. When the antrum was opened, the membrane was found to be ⅛ inch (0.32 cm.) thick and tucked up in edematous folds. The brown blood clot that was removed filled half the antrum. Since the operation there has been no further bleeding.

DR. H. P. MOSHER: Was the bleeding from the polyp?

DR. P. J. ROSS: There was a blood clot in the antrum. No bleeding point was seen in the antrum. There may have been bleeding from the polyp into the antrum. The nose was never packed.

DR. P. J. ROSS: A section of the polyp was examined.

DR. F. J. POLLOCK: This section of the polyp which was removed from the middle meatus reveals what is apparently a tumor mass. The cells in the center of the mass are large and pale-staining, and toward the periphery they form large spaces, most of which are filled with blood; there are a great many of these spaces. The appearance of the whole section is compatible with the diagnosis of hemangioendothelioma.

This section from the lining of the antrum shows a thickened, edematous mucous membrane, with the epithelium somewhat papillated. There is an infiltration by round cells, which is compatible with chronic inflammation. There are several areas of hemorrhage throughout the section, which may have been traumatic.

DR. D. C. SMYTH: Is a hemangio-endothelioma a benign tumor?

DR. F. J. POLLOCK: Yes, it is a tumor of the endothelium of the blood vessel and is benign.

DR. H. P. MOSHER: Is it worth while to use radium in these cases?

DR. D. C. SMYTH: I should think so. Why is it that hemangio-endotheliomas are malignant in the lung?

DR. F. J. POLLOCK: Do they metastasize?

DR. D. C. SMYTH: I don't know.

DR. F. J. POLLOCK: Hemangio-endotheliomas in the lung probably become malignant by invasion. They are probably benign microscopically, but are clinically malignant, owing to their inaccessible position in the lung, which does not permit their complete removal.

SIMPLE MASTOIDITIS WITH COMPLICATIONS. DR. E. M. HOLMES and DR. H. P. CAHILL.

A man, aged 48, had pain on the left side of the head. He had had headaches and deafness for six weeks. Paracentesis was performed several times at another hospital, with serous discharge. On admission, the patient complained that he had had postaural tenderness for the past five weeks. His memory was poor, and he had slow speech. For the past ten years he had been receiving treatment for tertiary syphilis. The right ear was normal. The left ear showed markedly decreased hearing and a thickened drum. A simple operation was performed on the mastoid, and pus was found under pressure in the cells and antrum. The bone over the dura was soft. The patient had marked pains throughout his head, without localization. He also had an abscess that extended to the neck, and a second operation was performed. Dr. Cahill explored beneath the mastoid to the digastric groove and found pus. Roentgenograms were taken after the injection of poppy-seed oil 40 per cent.

NOTE.—The fistulous opening into the neck persisted, and the postaural wound continued to drain. A third operation was decided on; the mastoid tip was removed, and the opening which Dr. Cahill found extending down toward the jugular bulb was reopened. There were no symptoms at this time to indicate anything more than the local disturbance in the peribulbar region. There were no chills, headache or fever. The patient was discharged from the hospital two weeks after operation, and after several weeks of treatment in the outpatient department the postaural wound and the fistula into the neck closed.

A week later the patient had a convulsion at home with disturbance in speech. He was brought back to the hospital. Neurologic examination revealed paralysis of the right side and definite aphasia. No convulsions were noted while the patient was under study. A left temporal decompression was done, and the temporal lobe was exposed. No abscess was found, but Dr. Tobey felt that he had entered a cyst cavity, as a thin bloody fluid escaped through the trocar. He did not believe that he had entered the ventricle.

The patient improved steadily for eight days after this procedure; then he had a sudden rise in temperature to 104 F. and stiffness of the neck and rapidly became comatose. The cerebrospinal fluid pressure rose to 700 mm., with the Tobey-Ayer test, and the dynamics were essentially normal. Without anesthesia the temporosphenoidal wound was explored, but nothing was found. A cerebellar decompression was then done, and in the attempt to enter the cerebellum it was noted that the dura was extremely thin. In cutting through what was thought to be the dura of the cerebellum, it was found that the lateral sinus was entered. It being realized that there was complete thrombosis of the lateral sinus, the clot was pushed back

almost to the torcula, and no bleeding was encountered. The clot was removed as far down and back as possible. There was no free pus in the sinus, though the clot was softened and gelatinous in the area through the mastoid. The cerebellum was explored through the sinus wall, and nothing was found. The veins of the scalp were tremendously engorged. The bone was excessively hemorrhagic, indicating that the diploe as well was engorged. The minute vessels in the tissues of the scalp bled freely.

Autopsy was not performed. The opinion of the staff when the patient was presented seemed to be that he had a long-standing sinus thrombosis which was symptomless, that the clot had extended beyond the torcula producing marked intracranial pressure, and that the symptoms of intracranial complication suggesting a temporosphenoidal abscess were probably due to the venostasis resulting from the thrombosis.

DISCUSSION

DR. H. P. MOSHER: Dr. Cahill consulted me. I found an exenterated mastoid, with pus coming from a hole in the anterior part of the bowl. The question is: Where did it come from? From the pharyngomaxillary fossa? Poppy-seed oil was put in, which gave a shadow coming from the mastoid underneath the sphenoid sinus and into the top of the sphenomaxillary fossa.

DR. A. S. MACMILLAN: The poppy-seed oil was injected into the hole mentioned; here (pointing to film) is the area of increased density, and here is another area of increased density directly behind the ear; in the anteroposterior view in the orbit the area of increased density is here, and the area of increased density is at this point. There seems to be a continuity of tract through here. The roentgenograms taken the next day showed that the man has a glass eye. He shot himself some years ago. The wound of entry was on one side of the nose, and the path of the bullet extended backward into the mastoid region and zygomatic region. When the first roentgenograms were taken, the side of the face on which the path of the bullet was deflected was turned away. The plates are very hazy in outline, which leads us to believe that there is a tract of poppy-seed oil going into the pterygomaxillary fossa.

DR. H. P. MOSHER: Dr. MacMillan told me that as a lead bullet goes through bone it is pared off, leaving bits of lead in its path.

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Prof. Gunnar Holmgren, Karolinska Mediko Kirurgiska Institut, Stockholm, Sweden.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg, 143, Groningen, The Netherlands.
Place: Stockholm, Sweden. Time: September 2-5, 1934.

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. W. P. Wherry, 107 S. 17th St., Omaha.
Secretary: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Place: Cleveland. Time: June 11-15, 1934.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. M. Wheeler, 30 W. 59th St., New York.
President-Elect: Dr. Wells P. Eagleton, 15 Lombardy St., Newark, N. J.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Sept. 9-14, 1934.

American Bronchoscopic Society

President: Dr. W. F. Zinn, Medical Arts Bldg., Baltimore.
Secretary: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.

American Laryngological Association

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Secretary: Dr. W. V. Mullin, 9204 Euclid Ave., Cleveland.
Place: Cleveland. Time: June 7-9, 1934.

American Laryngological, Rhinological and Otological Society

President: Dr. J. W. Jervey, 101 Church St., Greenville, S. C.
Secretary: Dr. R. L. Loughran, Bridgewater, Conn.

SECTIONS:

Eastern.—Secretary: Dr. Vernon L. Bishop, Medical Arts Bldg., Rochester, N. Y.
Southern.—Secretary: Dr. Fletcher D. Woodward, P. O. Box, 1685, University, Va.
Middle.—Secretary: Dr. Frank J. Novak, Jr., 30 N. Michigan Ave., Chicago.
Mid-Western.—Secretary: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis.
Western.—Secretary: Dr. Ralph F. Davis, Mayer Bldg., Portland, Ore.

American Otological Society

President: Dr. John R. Page, 127 E. 62nd St., New York.
Secretary: Dr. Thomas J. Harris, 104 E. Fortieth St., New York.
Place: Atlantic City. Time: 1934.

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* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.